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A REPORT OF FIFTEEN PATIENTS WITH TRAUMATIC RUPTURE OF THE THORACIC AORTA

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THE INCREASING FREQUENCY of traumatic rupture of the thoracic aorta has paralleled the increasing speed of modern transportation. Death usually occurs from immediate exsanguination after aortic rupture, but 10 to 20 per cent of the injured survive long enough for reparative treatment, provided a diagnosis can be established. Survivors of the initial injury appear to fall into two groups: those in whom the hemorrhage is contained in a hematoma but who may die of delayed rupture of this hematoma within the first few weeks after injury, and those surviving the initial and delayed threat who develop a false aneurysm which may remain stable for months or years. This report concerns our experience with patients in both categories—7 seen soon after aortic rupture and 8 seen with an aneurysm months or years after injury.

PATIENTS SEEN SOON AFTER INJURY

Brief case histories are given to emphasize the following facts: that aortic transection may be present with remarkably few external signs of injury, that a wide mediastinum on the chest roentgenogram and a history of injury are the most helpful diagnostic aids, and that there is often time for the institution of reparative treatment.

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*John and Mary R. Markle Scholar.

TABLE I. FINDINGS IN 7 PATIENTS SEEN SHORTLY AFTER INJURY WITH TRAUMATIC RUPTURE OF AORTA

NO.	INITIALS	AGE (YR.)	SYMPTOMS	FRACTURE	ADMISSION BLOOD PRESSURE (MM. Hg)	CHEST ROENTGENOGRAM	COURSE	PATHOLOGIC FINDINGS
1	J. G.	61	Dyspnea, leg pain	Rt. hip dislocation; ribs—multiple, bilateral	64/48	Mediastinal widening	Death 4 hours after injury	Aortic transection one inch distal to left subclavian artery
2	D. R.	33	None	None	Normal	Mediastinal widening	Sudden death 12 hours after injury	Aortic transection distal to subclavian artery
3	G. M. K.	46	Chest pain, dyspnea, weakness	Ribs—multiple, bilateral	110/60	Mediastinal widening; left hemothorax	Recurrent dyspnea and hypotension; death 14 hours after injury	Transverse aortic laceration distal to subclavian artery
4	R. P.	24	Pain, extremities	Humerus, ulna, radius, femur	88/40	Mediastinal widening; left hemothorax	Aortic occlusion with paraplegia and anuria; death 24 hours after injury	Transverse aortic laceration one inch distal to subclavian artery
5	A. E.	41	Chest pain	Ribs—multiple, right; fibula	Normal	Mediastinal widening	Sudden death 21 days after injury	Transverse aortic laceration distal to subclavian artery
6	C. B.	23	Unconscious	Ribs—multiple, right	80/58	Mediastinal widening; left hemothorax	Chest pain and systolic murmur 22 days after injury; aortic injury diagnosed; surgically repaired 37 days after injury	Aortic transection with 10 cm. aneurysm distal to subclavian artery
7	S. M.	23	Chest pain	Left first rib	Normal; previously had 1,000 ml. blood for hypotension	Mediastinal widening	Operative death 4 days after injury	Aortic transection with ends separated 3 cm. and dissection of aortic wall

The first 5 cases are grouped according to the length of time between injury and death; the last 2 cases are the only ones in which definitive surgical therapy was attempted. Pertinent details are tabulated in Table I.

CASE 1.—J. G., a 61-year-old man, was admitted with dyspnea and pain in the chest and right hip at 9 P.M. on Dec. 24, 1958, 30 minutes after an automobile accident. He had a blood pressure of 64/48 mm. Hg, multiple rib fractures, and a posterior dislocation of the right hip. A roentgenogram of the chest showed a marked widening of the mediastinum. The blood pressure promptly rose to 120/65 mm. Hg with intravenous fluid therapy and remained unchanged until 12 midnight. The dislocated hip was reduced and a splint applied to the lower extremity. At this time he complained of severe chest pain and quickly developed respiratory and cardiac arrest from which he could not be resuscitated. Several liters of blood were found in the left pleural cavity. Autopsy showed transection of the aorta 1 inch distal to the left subclavian artery with extensive hemorrhage into the mediastinum and left pleural cavity. Multiple rib fractures were present bilaterally.

Comment: The chest roentgenogram was an important clue to his underlying fatal condition.

Fig. 1.—Case 2. Male, aged 33, auto accident, no obvious injuries; died suddenly at home 9 hours later. Autopsy findings: transection of aorta and mediastinal hematoma.

CASE 2.—D. R., a 33-year-old white man, was examined on Aug. 26, 1956, shortly after an automobile accident and was found to have small lacerations of the chin and a contusion of the left anterior chest wall. A chest roentgenogram was interpreted as showing no abnormality, although subsequent review showed widening of the mediastinum. He was discharged from the Emergency Department 2 hours after admission. Nine hours later he was found dead in his bed at home. An autopsy showed transection of the aorta immediately distal to the left subclavian artery (Fig. 1) with a large mediastinal hematoma and several hundred milliliters of blood in the left pleural cavity. There were no rib fractures.

Comment: This patient had a transection of the aorta with strikingly few external signs of serious injury. The widened mediastinum on the roentgenogram was the only clue to the diagnosis.

CASE 3.—G. K., a 46-year-old white man, was seen in the Emergency Department at Baltimore City Hospital on Feb. 28, 1958, ½ hour after an automobile accident. He complained of chest pain and weakness. Examination showed a blood pressure of 110/60 mm. Hg,

moderate dyspnea, and râles over the left anterior chest. A chest roentgenogram was interpreted as showing no abnormality. Four hours after admission he became cyanotic and hypotensive but quickly improved with oxygen therapy. A second roentgenogram showed fluid in the left pleural cavity. The patient was discharged at his own insistence but became hypotensive at 2 P.M. the same day and was again hospitalized at 4:30 P.M. with marked dyspnea and a blood pressure of 85/50 mm. Hg. A chest roentgenogram showed a left hemothorax. He died shortly thereafter.

Autopsy showed a laceration of the aorta one inch distal to the subclavian artery which involved three fourths of the circumference of the aorta (Fig. 2). The associated mediastinal hematoma had ruptured into the pleural cavity. Multiple rib fractures were present bilaterally.

Comment: The serial roentgenograms showed widening of the mediastinum and a progressive left hemothorax. There was ample time for treatment had the correct diagnosis been made.

Fig. 2.—Case 3. Male, aged 46, auto accident, progressive hemothorax and death 14 hours later. Autopsy findings: laceration of aorta distal to subclavian artery with mediastinal hemorrhage.

CASE 4.—R. P., a 24-year-old white man, was hospitalized Aug. 22, 1958, 2 hours after an automobile accident. The blood pressure initially was 88/40 mm. Hg but subsequently rose to 140/100 mm. Hg. There were fractures of the right humerus, ulna, pelvis, and femur. A chest roentgenogram was of unsatisfactory quality. Four hours after admission, under general anesthesia, the fracture of the humerus was débrided and reduced. Following operation the feet were cold and pulseless. Several hours later he became paraplegic and anuric. Dyspnea gradually became more severe. Rupture of the aorta was diagnosed and thoracotomy planned, but death occurred at 11 P.M. in the operating room before the incision was begun.

Autopsy showed the fractures previously described and an almost complete transection of the aorta just distal to the subclavian artery (Fig. 3). There was a large mediastinal hematoma and a hemothorax. The distal aortic lumen was occluded by compression from hematoma in the adventitia.

Comment: This patient is the only one we have seen in whom rupture was associated with occlusion of the aorta with resulting paraplegia and anuria.

CASE 5.—A. E., a 41-year-old white man, was hospitalized Aug. 13, 1955, because of pain in the chest and leg after an automobile accident. He had fractures of the ribs and

the right tibia and fibula. Chest roentgenograms showed mediastinal widening and multiple rib fractures on the right (Fig. 4). A cast was applied to the right leg, and he was discharged 20 days later after an uneventful hospital course. Two days thereafter he suddenly collapsed and died.

Autopsy showed a laceration of the inner curvature of the aorta distal to the left subclavian artery. This communicated with a false aneurysm, 5 cm. in diameter, which had ruptured into the pleural cavity.

Comment: The most important clue to the diagnosis in this patient was the chest roentgenogram. A subsequent examination would almost surely have

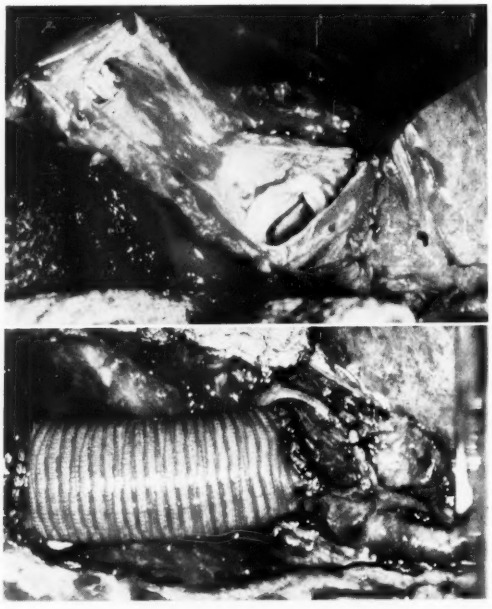
Fig. 3.—Case 4. Male, aged 24, auto accident caused multiple fractures in extremites; aortic occlusion 6 hours later, subsequent paraplegia and death. Autopsy findings: laceration of aorta distal to subclavian artery.

Fig. 4.—Case 5. Male, aged 41, auto accident, rib and extremity fractures, sudden death 21 days later from rupture of hematoma. Admission roentgenogram shows mediastinal widening and multiple rib fractures on the right.

showed further mediastinal enlargement. His survival for 22 days is the longest we have seen in the group of patients who died from delayed hemorrhage from the aorta.

CASE 6.—C. B., a 23-year-old man, was hospitalized Sept. 18, 1958, because of coma following an automobile accident. The blood pressure was 80/58 mm. Hg. A chest roentgenogram showed a moderate left hemothorax and fractures of the right seventh and eighth ribs. Subsequent roentgenograms on Sept. 19, 22, and 25 showed little change, except the last one did show some widening of the mediastinum to the left. He was discharged on October 1 but was readmitted on October 10 because of retrosternal pain. A systolic murmur was now audible over the precordium. A chest roentgenogram showed persistence of

A.



B.

Fig. 5.—Case 6. Male, aged 23, auto accident, rib fractures and left hemothorax, chest pain 22 days later, aorta repaired 35 days after injury.

A, Incised aneurysm showing the two ends of the aorta widely separated because of retraction of the intima.

B, Aneurysm has been excised and Dacron arterial prosthesis inserted. Left subclavian artery and recurrent laryngeal nerve are visible adjacent to the proximal anastomosis.

the mediastinal widening. A diagnosis of traumatic rupture of the aorta was made and thoracotomy was performed on October 23. A 5-cm. aneurysm was found distal to the subclavian artery. While a left atrial-femoral artery bypass was used to pump about 1,500 ml. of blood per minute, the aorta proximal and distal to the aneurysm was clamped and the aneurysm excised. A transection of the aorta was found with retraction of the ends for 3 cm. (Fig. 5, *A*). As an end-to-end anastomosis was not possible, a 7 cm. De Bakey Dacron prosthesis was inserted (Fig. 5, *B*). The postoperative course was uneventful; 1 year later he had no disability.

Comment: The widened mediastinum and the systolic murmur were the most important diagnostic findings.

CASE 7.—S. M., a 23-year-old man, was admitted to Walter Reed Army Hospital on Dec. 20, 1959, four days after an automobile accident; he had received 1,000 ml. of blood. There was a swelling in the left supraclavicular area, and a continuous murmur was audible over the precordium. A chest roentgenogram showed mediastinal widening (Fig. 6, *A*), and aortography demonstrated a wide irregularity in the region of the aortic isthmus (Fig. 6, *B*). At thoracotomy on December 21, after 900 ml. of blood had been removed from the left pleural cavity, a hematoma was found surrounding the thoracic aorta which extended proximally to the left carotid artery (Fig. 6, *C*). Left atrial-distal aortic bypass was used, the aorta clamped, and the hematoma incised. The aorta was found transected with retraction of the ends for about 3 cm. In addition, there was a dissection of the wall for a short distance in each end of the aorta extending in the proximal segment along the subclavian artery. A Teflon prosthesis was inserted but severe hemorrhage, which led to death, developed proximal to the graft, between the left carotid and subclavian arteries.

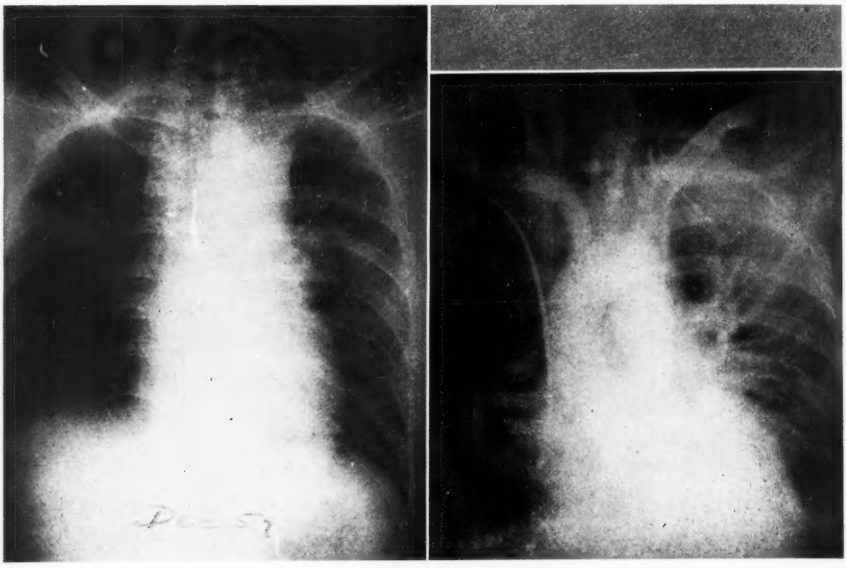
Comment: Dissection of the aorta extended proximal to the subclavian artery and caused the fatal hemorrhage. A wider excision of the injured area, extending proximally to the left carotid artery, might have allowed survival.

PATIENTS SEEN BECAUSE OF ANEURYSM MONTHS OR YEARS AFTER INJURY

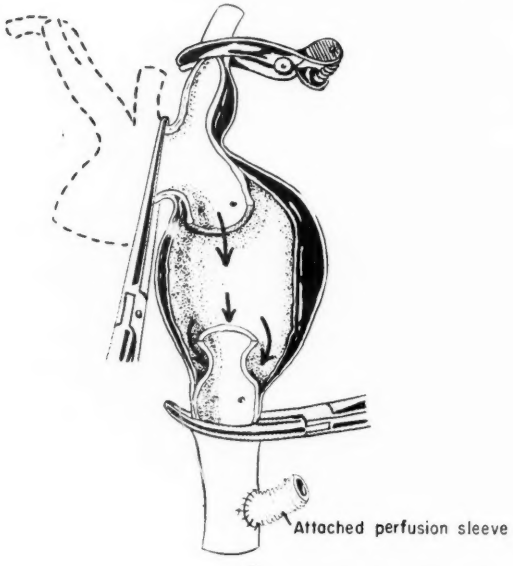
Eight patients were seen a long time after injury following discovery of a mediastinal mass on a chest roentgenogram (Cases 1, 37-43, Table II). An aortic laceration had not been suspected in any of this group in the period of recovery following the trauma. The data from the 8 patients are summarized in Table II and compared with similar thoracic aneurysms reported by others. Seven of the 8 were injured in an automobile accident, the remaining patient having been kicked by a calf over 37 years before. All of the aneurysms arose distal to the left subclavian artery. Three patients were asymptomatic, 3 had chest pain, 1 had a slight cough, and 1 had severe dyspnea from a large aneurysm which had previously been treated with a pneumonectomy because of atelectasis of the lung. A tangential excision was performed on 1 patient treated in 1952; all other patients were treated by excision and replacement with a plastic prosthesis, while the distal circulation was supported with left atrial-femoral artery bypass. The only patient treated unsuccessfully is described in detail because of his unusual course.

CASE REPORT.—W. N., a 43-year-old man (Case 37, Table II), was in an automobile accident in 1941 following which he had several fractured ribs and was unconscious for 14 hours. Periodic chest roentgenograms in military service were reported as negative until 3 years before admission when a mediastinal mass was seen. Eighteen months later dyspnea began and gradually increased to a severe degree. At thoracotomy in December, 1957, at Walter Reed Army Hospital, a huge aneurysm was found involving the aortic arch. It was deemed unwise to attempt its removal at this time as left atrial-femoral artery bypass equipment was not available. A pneumonectomy was performed because of atelectasis of the left lung. At re-operation on Jan. 13, 1958, the aneurysm was found encased in a dense pleural fibrosis, and attempted mobilization resulted in fatal hemorrhage. The aneurysm originated from a small opening in the aortic wall distal to the left subclavian artery; the aorta was normal both proximal and distal to the point of rupture.

Comment: It is unusual that the aneurysm did not change for 14 years but then, over a 3-year period, enlarged to massive proportions.



A. *B.*



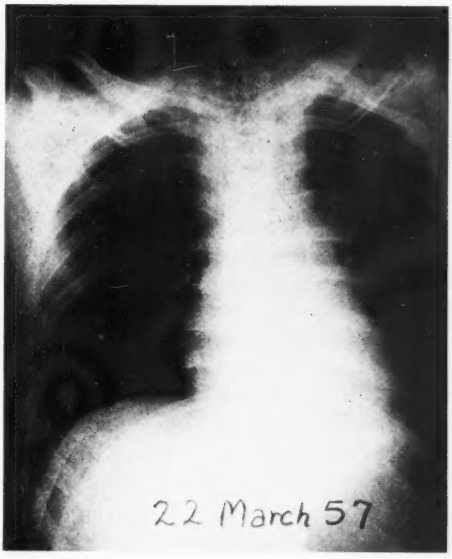
C.

Fig. 6.—Case 7. Male, aged 23, auto accident, ruptured aorta repaired 4 days after injury, operative death from hemorrhage.

A, Admission roentgenogram which shows mediastinal widening.

B, Aortography performed by injection of dye into right atrium. The area of rupture is shown as a localized irregular enlargement of the aorta distal to the subclavian artery.

C, Drawing which shows operative findings. The aorta was transected with formation of a mediastinal hematoma and dissection of the two ends of the aorta, extending in the proximal aorta into the subclavian artery. Repair was attempted after applying the clamps as shown.



A.

B.

Fig. 7.—Case 38. Male, aged 22, auto accident, fracture of pelvis, no thoracic symptoms, mediastinal mass found 2 years later, aneurysm excised.

A, Roentgenogram made after injury which shows widened mediastinum.

B, Aortogram made 2 years after injury showing localized aneurysm.

DISCUSSION

Frequency of Traumatic Injury of the Aorta.—Rupture of the aorta has long been recognized as a cause of immediate death after injury, but only a limited amount of data is available concerning patients who survive the initial injury. Parmley[29] in 1958 reviewed the course of patients dying from hemorrhage a short time after aortic rupture and reported 171 patients from the Armed Forces Institute of Pathology, 20 per cent of whom survived longer than 30 minutes after injury.

A limited experience has likewise been reported with patients with traumatic thoracic aneurysm. Reports of only 13 previously treated patients were found by Gerbode[14] in 1957 when he described experiences with 4 patients in whom the left atrial-femoral artery bypass technique was used. Descriptive data from 36 traumatic aneurysms reported since 1952, when excision of the aneurysm was first used for treatment, and the 7 new patients described in this report are shown in Table II. Ten additional patients referred to by De Bakey and Cooley,[10] 5 by Parmley,[29] and 1 each by Bosher[4] and Weisel[20] are not included because of the lack of detailed description. Much of the data in Table II is from the previous report by Gerbode, but additional patients are included who have been observed but not operated upon. The total group of 60 patients represents a fairly complete review of cases described since 1952. This small number indicates the comparative rarity of traumatic thoracic aneurysms and emphasizes the fact that clinical experience with such problems is limited.

Etiology.—Fourteen of our 15 patients were injured in an automobile accident, while 20 of the 28 remaining patients listed in Table II were similarly injured, this being the most common cause of an aortic laceration. Uncommon causes include a fall from a height,[27] direct crushing blows to the chest,[27] or a sudden rise in intravascular pressure, such as that caused by being covered with falling earth in a mine cave-in.[29]

The stress resulting from unequal rates of horizontal deceleration is thought to be the basic cause of aortic rupture in automobile accidents. The difference in rate of deceleration of the mobile descending thoracic aorta and the relatively fixed arch of the aorta strains the junction of the two areas at the aortic isthmus where the ligamentum arteriosum is attached. This mechanism has been suggested by several authors.[6, 14, 30, 31] One of the more detailed studies of this subject was made by Hass[18] in an evaluation of death from airplane accidents. Vertical deceleration, as in falls from a height, results in stress in a different area and more frequently causes aortic laceration just above the aortic valve.[6]

Pathology.—The aortic injury is usually just distal to the left subclavian artery. This was the location in all 15 of our patients, in 40 of 72 cases reported by Strassmann,[38] in 24 of 38 patients living over 30 minutes after injury reported by Parmley,[29] and in almost all the individual case reports in the literature.[6, 11, 12, 19, 24, 28, 29, 30, 33, 40] The next most common site of injury is the ascending aorta. Injuries of the aortic arch, descending thoracic aorta, or abdominal aorta are rarely seen and are of less clinical significance because they usually result from more severe trauma causing immediate death.[29] The aneurysm was located distal to the subclavian artery in 38 of the 43 patients listed

in Table II. In the other 5 cases, the descending thoracic aorta was involved in 2 and the ascending aorta in 3; in 2 of the latter there was a dissecting aneurysm.

The aortic tear is a transverse one which may extend through the intima and media with preservation of the adventitia, or it may be through all layers with retraction of the two ends. Part or all of the circumference of the aorta may be involved. Four of our 7 patients seen shortly after injury and 9 of the 38 patients reported by Parmley had a complete circumferential transection of the aorta; the others had incomplete lacerations. Patients who do not exsanguinate immediately are probably those in whom the adventitia remains intact for a short period of time. It has been estimated that the adventitia provides about 60 per cent of the tensile strength of the aorta.[11] The tear probably begins in the intima and proceeds outward, as Cammack[6] has suggested; this is an important consideration because the degree of injury may be greater within the lumen than can be detected by external examination.

It is surprising that a dissecting aneurysm rarely develops after rupture of the aortic intima and media. This infrequent occurrence may be due to the absence of disease of the aortic wall, as dissecting aneurysms are thought usually to result from disease of the aortic media. Five patients who did develop dissecting aneurysms lived a few weeks or months before dying from rupture of the aneurysm or cardiac failure.[26, 27, 31, 32]

If the aortic tear does not result in immediate death from hemorrhage, a secondary fatal hemorrhage may occur in the next few weeks. After this time hemorrhage rarely occurs, a feature that contrasts markedly with aneurysms of syphilitic or arteriosclerotic origin. Deaths in all of our patients and most deaths reported by others from delayed rupture of the aorta have occurred within 4 weeks after injury. Only a few exceptions to this course have been recorded. Parmley described a patient dying from hemorrhage from a laceration in the ascending aorta 50 days after injury.[29] A patient reported by Bradford and Johnston[5] died from hemorrhage 81 days after laceration of the aorta above the diaphragm. Jay and French[21] reported 1 case of survival for 2 months before erosion of the left main bronchus resulted in fatal hemorrhage.

It was impressive in the cases reviewed (Table II) that no deaths were found from rupture of a traumatic aneurysm over a few months in duration. Such cases may simply not have been reported. Another possibility is that since aneurysms of a few years' duration are usually calcified, they could easily be misdiagnosed at autopsy as arteriosclerotic or syphilitic. The proximity of traumatic aneurysms to the left main bronchus also aids detection before a large aneurysm develops because symptoms soon arise from compression of the bronchus and the adjacent recurrent laryngeal nerve. Several enlarging aneurysms (Table II) have been discovered and treated because of the appearance of these symptoms.

The rate of growth of traumatic aneurysms has varied, but most have not enlarged for many years. Four of our 8 patients had some enlargement before operation, but the degree of growth was slight in 2 of these (Cases 1 and 42, Table II). Case 37 furnished an impressive example of delayed growth; after

TABLE II. TRAUMATIC THORACIC ANEURYSMS

NO.	AUTHOR DATE	AGE (YR.)	CAUSE	DURATION (YR.)	OPERATION						OPERATION NOT DONE (SUBSEQUENT COURSE)
					LOCATION	SYMPTOMS	OTHER FINDINGS	SIZE OF ANEURYSM (GREATEST DIAMETER, CM.)	OPERATIVE PROCEDURE	RESULT	
1	Bahnson[3] 1952	49	Auto accident	2	Ligamentum arteriosum area	Hoarseness	Enlarging mass	9	Tangential excision	Good, 8 yr. postop.	
2	Hollingsworth et al.[19] 1952	29	Auto accident	2	Ligamentum arteriosum area	None		7	Covered with cellophane	No change 2 yr. later	
3	Hollingsworth et al.[19] 1952	27	Auto accident	7	Ligamentum arteriosum area	None		5			Observation 5 years; asymptomatic
4	Hollingsworth et al.[19] 1952	19	Auto accident	1	Ligamentum arteriosum area	None					Observation 6 months; asymptomatic; enlarging mass
5	Johnston et al.[22] 1953	66	Auto accident	3	Ligamentum arteriosum area	Dyspnea	Left vocal cord paralysis	10	Tangential excision	Good	
6	Stranahan et al.[37] 1953	25	Auto accident	5	Ligamentum arteriosum area	None	30 per cent compression left bronchus	7	Excision; homograft	Good	
7	Hardin[17] 1953	32	Struck by coal conveyor	10	Ligamentum arteriosum area	None		8	Excision; homograft	Died; ventricular fibrillation	
8	Goyette et al.[15] 1953	23	Auto accident	7 mo.	Ligamentum arteriosum area	None	Enlarging mass	9	Lateral excision; polyethylene wrap	Fatal rupture 3 mo. postoperative	
9	Goyette et al.[15] 1953	28	Auto accident	8	Ligamentum arteriosum area			6-8			Observation several months
10	Goyette et al.[15] 1953	22	Auto accident	3	Ligamentum arteriosum area			4-6			Observation several months
11	Cooley et al.[7] 1954	31	Crushing injury	9	Ligamentum arteriosum area	None (1st operation) pain;		6	Tangential excision; (1st operation) Excision;	Aneurysm recurred; re-operated upon	

	et al.[13] 1953		accident		teriosum area					several months
11	Cooley et al.[7] 1954	31	Crushing injury	9	Ligamentum arteriosum area	None (1st operation) pain; hoarseness; dyspnea (2nd operation)		6	Tangential excision; (1st operation) Excision; homograft (2nd operation)	Aneurysm recurred; re-operated upon Good
12	Julian et al.[23] 1954	22	Auto accident	5	Ligamentum arteriosum area	None		Not recorded	Excision; homograft	Good
13	Ware et al.[39] 1954	19	Auto accident	9 wk.	Ligamentum arteriosum area	Slight dyspnea and pain	Enlarging mass	12	Excision; homograft	Died; esophagopleural fistula
14	Adams[2] 1955	30	Crushing injury	1	Ligamentum arteriosum area	None		Not recorded	Excision, homograft	Good
15	Storey et al.[36] 1955	35	Fall	4	Ligamentum arteriosum area	None		10	Excision; homograft	Good
16.	Hardin[17] 1955	31	Land mine	12	Descending thoracic aorta	Dyspnea; chest pain		Multiple: 6; 14	Excision; Orlon graft	Good
17	Cooley and De Bakey[8] 1956	50	Horse kick	20	Ascending aorta	Chest pain			Excision; homograft	Good
18	Holmes and Netterville[20] 1956	24	Auto accident	3	Ligamentum arteriosum area	None		8	Polyethylene wrap	No change 14 mo. later
19	Gerbode et al.[14] 1956	18	Auto accident	2	Ligamentum arteriosum area	None		7	Excision; homograft	Died; hemorrhage
20	Gerbode et al.[14] 1956	25	Auto accident	13	Ligamentum arteriosum area	Fever, cough, dyspnea, pain	50 per cent occlusion left bronchus	10	Excision; homograft	Good
21	Gerbode et al.[14] 1957	19	Auto accident	3	Ligamentum arteriosum area	None		7	Excision; homograft	Good
22	Gerbode et al.[14] 1957	23	Auto accident	2 mo.	Ligamentum arteriosum area	Pain; dyspnea		12	Excision; homograft	Good
23	Steinberg and Evans[35] 1957	29	Auto accident	4	Ligamentum arteriosum area	None		8	Excision; homograft	Died

TABLE II—CONT'D

NO.	AUTHOR DATE	AGE (YR.)	CAUSE	DURATION (YR.)	OPERATION						OPERATION NOT DONE (SUBSEQUENT COURSE)
					LOCATION	SYMPTOMS	OTHER FINDINGS	SIZE OF ANEURYSM (GREATEST DIAMETER, CM.)	OPERATIVE PROCEDURE	RESULT	
24	Steinberg and Evans[35] 1957	25	Auto accident at 4 years of age	21	Ligamentum arteriosum area			7			Observation 9 yr.; slight back pain
25	Steinberg and Evans[35] 1957	49	Auto accident	28	Ligamentum arteriosum area			6			Observation 1 yr.; asymptomatic
26	Steinberg and Evans[35] 1957	47	Auto accident	2	Ligamentum arteriosum area			9			Observation 2 yr.; asymptomatic
27	Steinberg and Evans[35] 1957	44	Auto accident	21	Ligamentum arteriosum area			6			Observation 1 yr.; asymptomatic
28	Mazzitello[27] 1957	47	Blunt injury	8 mo.	Ascending aorta; dissecting						Died from aortic insufficiency and congestive failure
29	Mazzitello[27] 1957	23	Auto accident	3 mo.	Ascending aorta; dissecting						Died from myocardial infarction and congestive failure
30	King and Shumacker[25] 1957	30	Auto accident	10	Ligamentum arteriosum area	None		10	Excision; nylon graft	Good	
31	Eiseman and Rainer[11] 1958	42	Auto accident	16	Ligamentum arteriosum area			10			Observation 15 yr.; asymptomatic
32	Eiseman and	24	Auto accident	6	Ligamentum arteriosum area			12			Observation 4 yr.; asymp-

	1958										asymptomatic
32	Eiseman and Rainer[11] 1958	24	Auto accident	6	Ligamentum arteriosum area			12			Observation 4 yr.; asymptomatic
33	Eiseman and Rainer[11] 1958	53	Auto accident	13	Descending thoracic aorta	Back pain	Enlarging mass	11	Excision; homograft	Good	
34	Eiseman and Rainer[11] 1958	63	Mule kick	47 ?	Descending thoracic aorta	None		12			Observation 9 yr.; enlarged in last 4 yr.
35	Parmley et al.[29] 1958	27	Auto accident	1	Descending thoracic aorta	Slight dyspnea; pain		8	Excision; homograft	Good	
36	Parmley et al.[29] 1958	20	Auto accident	3 mo.	Descending thoracic aorta	None		7	Excision; homograft	Good	
37	This report 1960	43	Auto accident	14	Origin distal to subclavian; involved aortic arch	Atelectasis; dyspnea; severe	Atelectasis; previous left lung Pneumonectomy	15	Attempted excision; hemorrhage	Died	
38	This report 1960	22	Auto accident	2	Ligamentum arteriosum area	None	Enlarging mass		Excision; Teflon graft	Good	
39	This report 1960	20	Auto accident	9 mo.	Ligamentum arteriosum area	Chest pain	Systolic murmur		Excision; Teflon graft	Good	
40	This report 1960	21	Auto accident	4 mo.	Ligamentum arteriosum area	None		5	Excision; Teflon graft	Good	
41	This report 1960	27	Auto accident	5	Ligamentum arteriosum area	Chest pain	Systolic murmur	10	Excision; Dacron graft	Good	
42	This report 1960	46	Auto accident	10	Ligamentum arteriosum area	None	Slight enlargement recent months		Excision; Teflon graft	Good	
43	This report 1960	52	Calf kick	37	Ligamentum arteriosum area	None	Attempted excision at previous thoracotomy unsuccessful	8	Excision; Teflon graft	Good	

a 14-year latent period, the aneurysm enlarged to massive proportions over a 3-year period. By contrast, the aneurysm in Case 43 may have been present for 37 years but was only 8 cm. in diameter when removed.

Seven patients in Table II (Cases 6, 8, 11, 13, 17, 20, and 31) had progressive enlargement of the aneurysm before excision. The aneurysms in 11 other patients have been observed for as long as 15 years with only one showing progressive enlargement. This patient (Case 34), whose aneurysm probably resulted from a kick by a mule 47 years earlier, had had progressive enlargement for 4 years, but operative therapy had been avoided at the time of the report because of a metastatic malignancy.

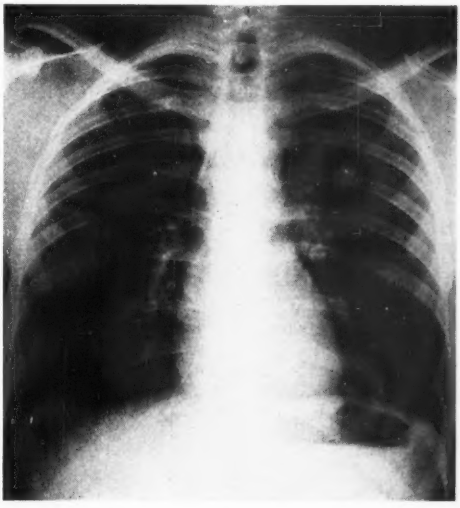
Steinberg[34] has emphasized the relative stability of traumatic aneurysms and presented 5 cases in which there was no enlargement over long periods of time, 1 presumably for 28 years. The eventual outcome of such aneurysms is unknown, but our patient (Case 37) with a fatal enlargement of his aneurysm starting 14 years after injury and the patient described by Eiseman (Case 34) with progressive enlargement starting 43 years after injury suggest that enlargement remains an ever present possibility.

Clinical Findings.—Dyspnea and chest pain were the most common symptoms in patients seen shortly after injury (Table I). These symptoms were of no diagnostic value, as they were no more prominent than expected with rib fractures which were present in 6 of the 7 patients. Fractures of the extremities were present in 3 patients; only 1 patient (Case 2) had no fractures. Two patients had shock of moderate degree, 2 had transient hypotension, and 3 had a normal blood pressure. Murmurs were heard in 2 patients, a continuous murmur in Case 7 and a systolic one in Case 6.

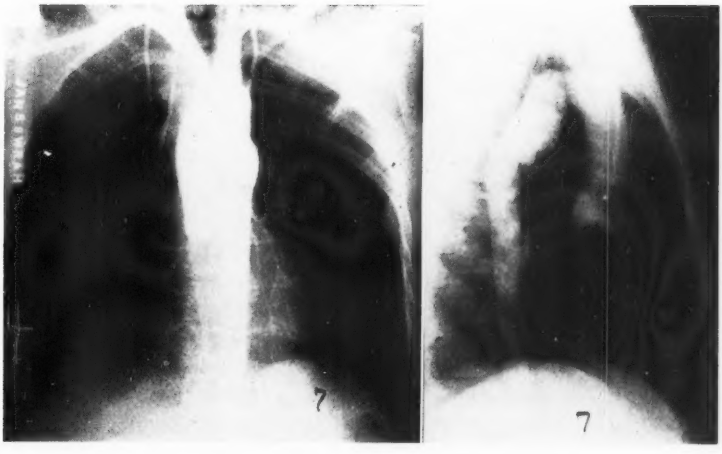
These nonspecific findings are similar to those reported by others. No diagnostic symptoms or signs were found by Parmley in his review of 57 previously reported and 38 new cases in which the patient lived over 30 minutes after injury. About one half the patients lost consciousness briefly after injury. One third had moderate hypotension, but persistent shock was seen only with recurrent hemothorax.[11, 12, 29, 30] Rib fractures were usually present, and extremities were fractured in about one half the patients. Murmurs were rarely described; no abnormality of the peripheral pulses was seen.

Two of our patients seen early after aortic rupture had no symptoms preceding sudden death (Cases 2, 5, Table I), 3 had recurrent hemothorax with progressive mediastinal widening, and 1 (Case 6) had increased chest pain 22 days after injury, investigation of which led to the correct diagnosis and subsequent surgical repair. The course of Case 4 was that of acute thoracic aortic occlusion with paraplegia and anuria; we found such an unusual course reported in only 1 other case in the literature.[11] The diagnosis was suspected in Case 7 because there was a wide mediastinum and an unusual continuous murmur. Aortography confirmed the diagnosis. Other reported patients have developed dyspnea, hoarseness, or dysphagia from compression of adjacent structures by the expanding mediastinal hematoma,[28, 33] and in some cases a fatal rupture was preceded by a sudden exertion or strain.[21, 24, 26]

After the hematoma becomes organized and an aneurysm forms, the patients are often asymptomatic, and the aneurysm is discovered as an incidental finding on a chest roentgenogram. If present, symptoms are likely to be hoarseness, or dyspnea resulting from pressure on the adjacent nerves or left main bronchus. Symptoms were present in only 16 of the 43 collected cases in Table II.



A.



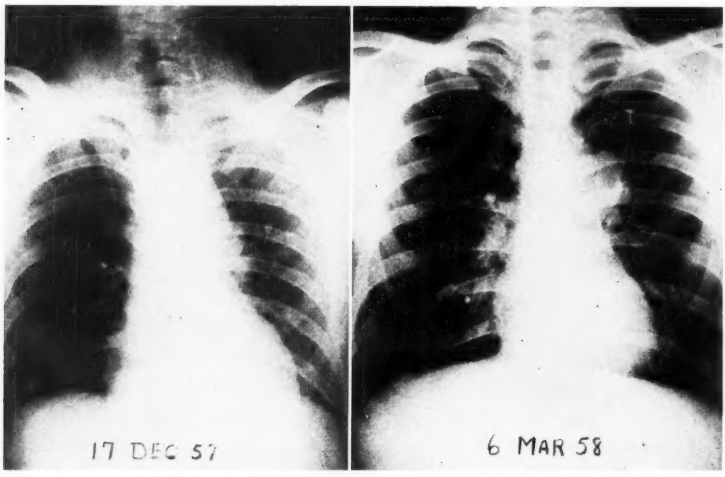
B.

Fig. 8.—Case 39. Male, aged 20, auto accident, ruptured spleen and diaphragm repaired, no aortic injury recognized; mediastinal mass noted 5 months later, aneurysm excised.

A, Roentgenogram made 5 months after injury showing small mediastinal mass below aortic knob.

B, Aortogram which demonstrates localized aneurysm.

Roentgenologic Findings.—Widening of the upper mediastinum was seen on the chest roentgenogram in all 7 patients observed soon after injury, and a hemothorax was seen in 3 (Table I, Figs. 4, 6*A*, 7*A*, 9*A*). The widened mediastinum is due to extravasation of blood from the aortic laceration and has been emphasized by several authors as diagnostic of mediastinal hemorrhage and suggestive of an aortic laceration.[11, 19, 35, 40] Unfortunately the widening is often not extensive and may be overlooked. The correct diagnosis was suggested in only 2 of our 7 patients on the initial roentgenographic interpretation.



A. *B.*

Fig. 9.—Case 40. Male, aged 21, auto accident, no obvious thoracic injury, mediastinal mass found 3 months later, aneurysm excised.
A, Roentgenogram made shortly after injury shows mediastinal widening.
B, Roentgenogram made 3 months later shows localized mediastinal mass.

Traumatic aneurysms several weeks after injury are visible as a localized mass in the left mediastinum near the aortic knob (Figs. 8, *A* and 9, *B*). The transition of the roentgenographic findings from a widened mediastinum shortly after injury to a localized mass a few weeks later has been well illustrated by Hollingsworth[19] (Fig. 9, *A* and *B*).

Diagnosis.—Awareness of the fact that aortic rupture can occur with any chest injury is the first step in diagnosis. Any degree of widening of the mediastinum on chest roentgenogram should then be carefully investigated, an extensive or progressive widening without symptoms probably being an adequate indication for immediate thoracotomy. This management would have provided the only chance of survival for Cases 1 and 5 (Table I). Slight degrees of widening are not diagnostic because mediastinal hemorrhage may result from causes other than aortic lacerations. Wyman[40] followed 1 patient with serial roentgenograms who had significant widening of the mediastinum soon after injury. As the mediastinal shadow became normal in a few weeks, the widening after injury was thought to have resulted from mediastinal hemorrhage from injured blood vessels other than the aorta. We performed a

thoracotomy on 1 patient with a widened mediastinum shortly after injury and found a tortuous aorta without mediastinal hemorrhage. Recurrent hemothorax, dyspnea, dysphagia, or hoarseness may suggest the diagnosis. When the patient's condition permits, aortography may be useful. The diagnosis was readily made in Case 7 by finding a localized irregular widening of the aortic isthmus (Fig. 6, *B*). Emergency aortography should be used more frequently for such problems.

Diagnosis of an aneurysm, once it has formed, is usually not difficult in the presence of a mediastinal mass in the region of the aortic isthmus. Aortography readily confirms the diagnosis (Figs. 7*B*, 8*B*, 10).

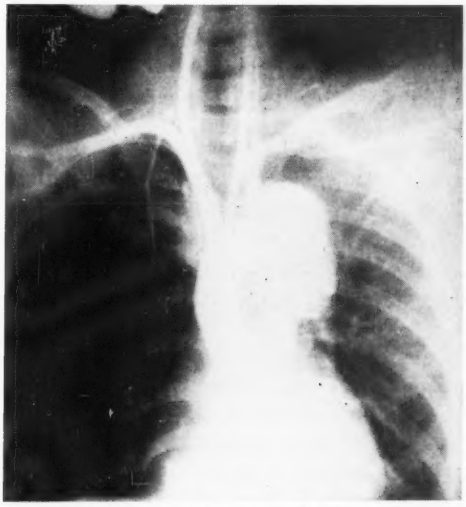


Fig. 10.—Case 41. Female, aged 27, auto accident, asymptomatic mediastinal mass noted 6 months later; chest and back pain 4 years later, aneurysm excised. Aortogram made 5 years after injury shows large localized aneurysm.

Surgical Considerations.—The consistent location of the aortic injury as a transverse tear just distal to the origin of the left subclavian artery makes surgical repair feasible if the aorta can be isolated proximal and distal to this area. When the acutely ruptured aorta is explored, it must be isolated at a point removed from the hematoma. Approaching the proximal aorta by opening the pericardium in Case 7 allowed us to encircle the aorta proximal to the left subclavian artery without entering the mediastinal hematoma. This seems an important safety measure.

Several nearly successful cases have been reported which emphasize the technical problems that may be encountered. Murdock[28] explored 1 patient who suddenly developed dyspnea and dysphagia 10 days after injury, but fatal hemorrhage occurred before the aorta could be isolated. Ellis's[12] patient was explored for an enlarging hemothorax 9 days after injury, but paraplegia and death followed 96 minutes of aortic occlusion while a homograft was inserted. Shumacker and King[33] explored a patient, 25 days after injury, who had occlusion of the left main bronchus and perforation of the esophagus from an

expanding hematoma. The aorta was successfully repaired, but an esophageal fistula developed and caused death 7 weeks later. The gravity of esophageal injury is emphasized by the report of 1 other patient[39] in whom the esophagus was injured during excision of an aneurysm 9 weeks after injury; an esophageal fistula developed and caused fatal hemorrhage from the aortic suture line 7 days after operation.

Eiseman[11] found diffuse ecchymosis surrounding the entire thoracic aorta in a patient explored a few hours after injury because of marked widening of the mediastinum. The laceration could not be found, but an aneurysm subsequently developed, and death resulted from attempted excision 6 weeks later. Passaron and Pace[30] reported the suture of a 3 mm. adventitial perforation in a patient explored 8 hours after injury for an expanding mediastinal hematoma. Subsequent aortography showed an aneurysm developing on the posterior aortic wall opposite the area of repair. Both the foregoing reports emphasize that the most extensive injury is on the intimal surface and can be found only by opening the aorta, as suggested by Eiseman.[11] Although uncommon, aortic dissection of the adjacent ends may occur and probably was responsible for the fatal hemorrhage in our Case 7; excision or repair of the area of dissection may be necessary.

The operative management of all lesions of the descending thoracic aorta has been improved by the use of left atrial-femoral artery bypass described by Cooley[9] and Gerbode.[14] The technique is an uncomplicated one which adds little to the operative risk, and equipment for it should be available when circumstances permit, because aortic occlusion for longer than 20 minutes is associated with an increasing risk of paraplegia.[1] All but 1 of our patients were operated upon in this manner. A woven Teflon prosthesis is preferred over knitted Dacron because, when heparin is used, blood loss is greater through the larger interstices of the knitted materials. As in the treatment of other aneurysms, the entire sac should be removed if possible. However, the dense fibrous tissue surrounding the aneurysm may make dissection difficult and also increase blood loss when heparin is given before instituting left atrial-femoral artery bypass. We have preferred to limit initial dissection to mobilization of the adjacent aorta, after which the aorta is occluded, the aneurysm incised, and the aortic prosthetic graft inserted. After the heparin has been neutralized, the wall of the aneurysm can be readily dissected from adjacent tissues. Injury to the esophagus should be carefully avoided; both patients reviewed in this report with esophageal injury, 1 from erosion[33] and 1 from surgical injury,[39] ultimately died from complications of an esophageal fistula.

The good results with present surgical techniques and the possibility of expansion of an aneurysm after a latent period of many years both indicate that excision is the preferred treatment, even if no symptoms are present. However, the almost negligible risk of rupture of small aneurysms and the prompt appearance of symptoms when such aneurysms enlarge indicate that excisional therapy for small asymptomatic aneurysms is not urgent, and such lesions may be safely followed with periodic observation if age or coincidental disease makes surgical excision hazardous.

SUMMARY

Experiences with 7 patients with traumatic rupture of the aorta treated shortly after injury and 8 patients wtih traumatic aneurysms treated months or years after injury have been described. Only 1 of the 7 patients seen soon after traumatic rupture survived, chiefly because the diagnosis was either not made or made too late for treatment to be successful. The aortic injury was surgically repaired in 2 patients with a successful outcome in 1. All of the lacerations resulted from automobile accidents, and all were located just distal to the left subclavian artery, an area readily accessible to surgical repair. Symptoms and signs usually originated from associated rib fractures and were of no diagnostic significance. A widened mediastinum was seen on chest roentgenogram in all 7 patients; this finding was of great diagnostic significance and, in 2 patients, was the only abnormality recognized. Emergency aortography confirmed the diagnosis in 1 patient and should probably be used more frequently.

Other reports reviewed show that 10 to 15 per cent of the patients with an aortic laceration survive for short periods of time only to die from hemorrhage in the succeeding weeks. The usual location of the injury in the aorta distal to the origin of the left subclavian artery makes surgical repair feasible, but to date this has seldom been accomplished, chiefly because of inaccuracy or delay in diagnosis.

Patients living more than a few weeks after aortic rupture develop an aneurysm but rarely hemorrhage from its rupture. Eight such patients were treated, 7 of whom survived. The relative infrequency of such aneurysms is indicated by finding only 52 similar lesions reported in recent years. The growth rate was different from aneurysms from other causes; a few enlarged soon after injury, some remained small and asymptomatic for indefinite periods, and some gradually enlarged after a latent period of many years. Dyspnea, dysphagia, hoarseness, and pain appeared when the aneurysm enlarged and compressed adjacent structures. The preferred treatment is excision and replacement with a plastic prosthesis, the distal circulation being supported with left atrial-femoral artery bypass while the aorta is occluded. Results have been good and the mortality rate low. Small asymptomatic aneurysms may be safely observed if there is additional disease present which makes operation hazardous.

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(For Discussion, see page 95)

SURGICAL MANAGEMENT OF PENETRATING INJURIES OF THE ASCENDING AORTA AND AORTIC ARCH

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INJURIES which produce a penetrating or lacerated wound of the aorta are usually fatal. The chance for survival is better if the intrapericardial aorta is injured because of the tamponading effect of the pericardial sac. The successful repair of a wound of the aorta within the pericardium was reported by Dshanelidze[1] of Russia in 1922, by Blalock[2] in 1934, and by Elkin[3, 4] in 1941 and 1944. In 1950, Beattie and Greer[5] removed a 6 cm. segment of knife blade from the aorta within the pericardium and closed a laceration in the aortic wall which was 14 mm. in length. The operation was performed 48 hours after injury and the patient recovered. In 1956, Perkins and Elchos[6] successfully closed a 2 cm. lacerated wound of the aortic arch at the base of the innominate artery; operation was performed about 2 hours after injury. They were unable to find a previous report of the successful early repair of an extrapericardial laceration of the ascending aorta. Proctor[7] and McCook[8] have each successfully repaired a traumatic arteriovenous fistula between the aortic arch and the left innominate vein.

More complicated injuries involving the aorta have been reported in recent years. Gerbode[9] encountered a patient in whom a bullet wound produced an aortico-right atrial fistula and aortic insufficiency. The patient underwent operation but the lesion was not repaired; the patient subsequently died of heart failure. Gillanders[10] reported a traumatic aorticopulmonary fistula caused by a needle. The defect, which measured 3.7 cm. in length, was immediately above the ventricular septum and could not be closed. King and Shumacker[11] have repaired an aortico-right ventricular fistula which resulted from a bullet wound; the patient recovered. Morris and his associates[12] reported two instances of aortico-right ventricular fistula resulting from stab wounds; both defects were successfully repaired.

We have observed and treated 3 patients with injuries of the ascending aorta and the aortic arch. These cases form the basis of this report.

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CASE REPORTS

CASE 1.—B. T., a 36-year-old Negro woman, was admitted to the George W. Hubbard Hospital on Dec. 25, 1952, about 30 minutes after receiving a bullet wound of the left, upper, anterior chest. The patient was dyspneic, cyanotic, and had distended neck veins. Blood pressure was 86/38 mm. Hg; the pulse rate was 140 per minute. There was a penetrating wound in the first left intercostal space near the sternal border. Heart sounds were distant; breath sounds were normal bilaterally. A diagnosis of acute pericardial tamponade was made and the pericardial sac was aspirated of 55 and 35 c.c. of dark blood on two occasions within a short period of time, following which the patient's general condition improved markedly. A portable chest roentgenogram showed widening of the mediastinal and cardiac

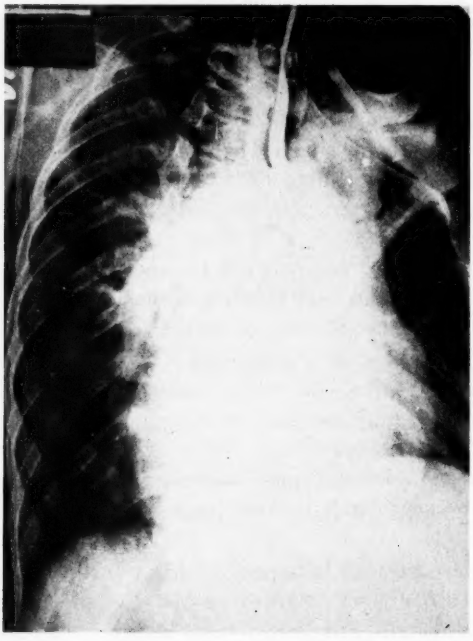


Fig. 1.—Case 1. Retrograde aortogram shows simultaneous filling of aorta and left pulmonary artery.

shadows with some diffuse haziness over the left lung field, but no pneumothorax. She was given 1,000 c.c. of whole blood shortly after admission and the blood pressure became stable at 100/60 mm. Hg. During the next few days she gradually improved and the cardiac silhouette diminished in size. On Jan. 12, 1953, a blowing systolic murmur was heard over the pulmonic area. She was discharged from the hospital on Jan. 20, 1953.

She was again admitted to the hospital on Feb. 2, 1953. During the interval she noted the development of dyspnea and ankle edema. Examination again revealed a blowing systolic murmur over the pulmonic area. The blood pressure was 94/54 mm. Hg; the venous pressure was 197 mm. of saline. An electrocardiogram was considered to be within normal limits. She was placed on a salt free diet and discharged from the hospital on Feb. 17, 1953, to be followed in the cardiac clinic. On March 6, 1953, there was no sign of congestive failure. For the first time a continuous harsh murmur and thrill at the base of the heart and accentuation of the pulmonic second sound were noted.

She was again admitted to the hospital on April 25, 1953, for further study. She was slightly dyspneic but was not in cardiac failure. The blood pressure was 120/60 mm. Hg.

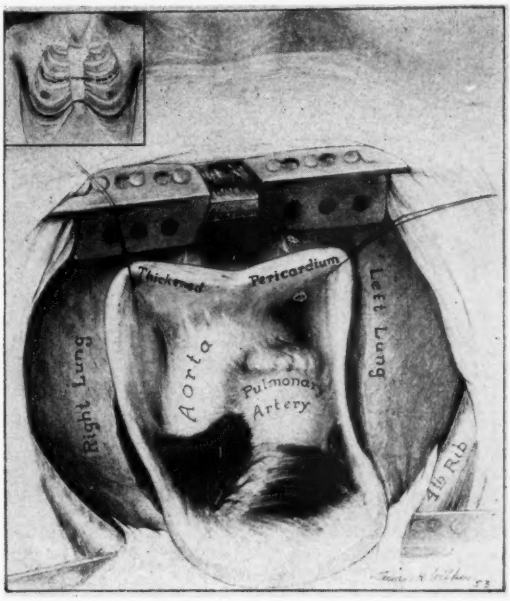


Fig. 2.—Case 1. Drawing which depicts the fistula between the aortic arch and the pulmonary artery.

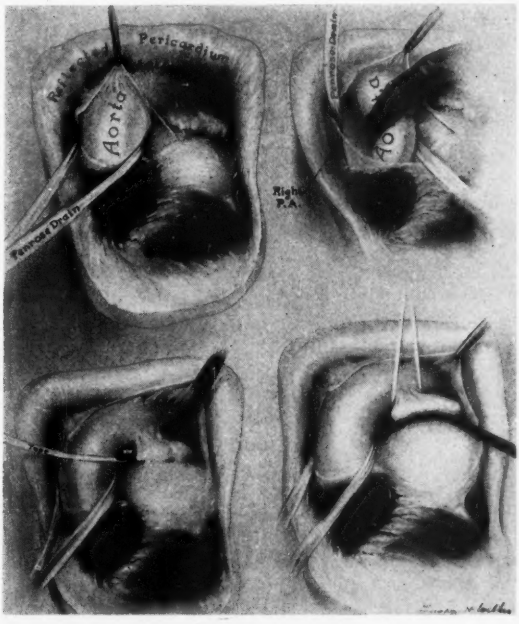


Fig. 3.—Case 1. Dissection of aortic arch and the aorticopulmonary fistula.

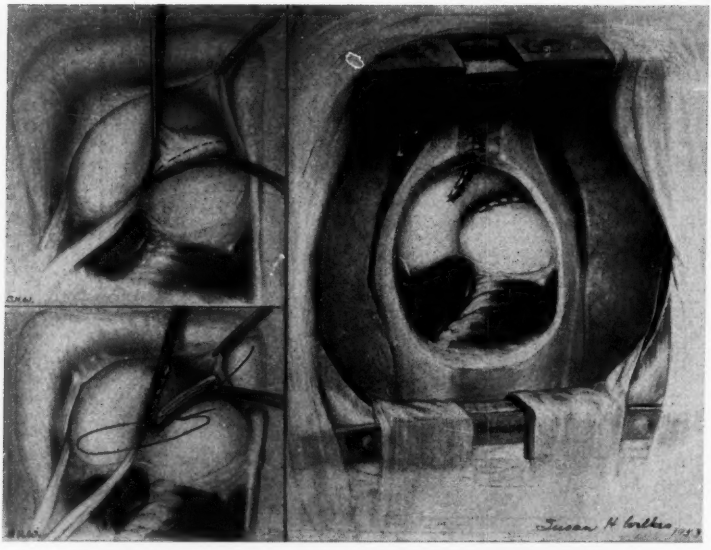
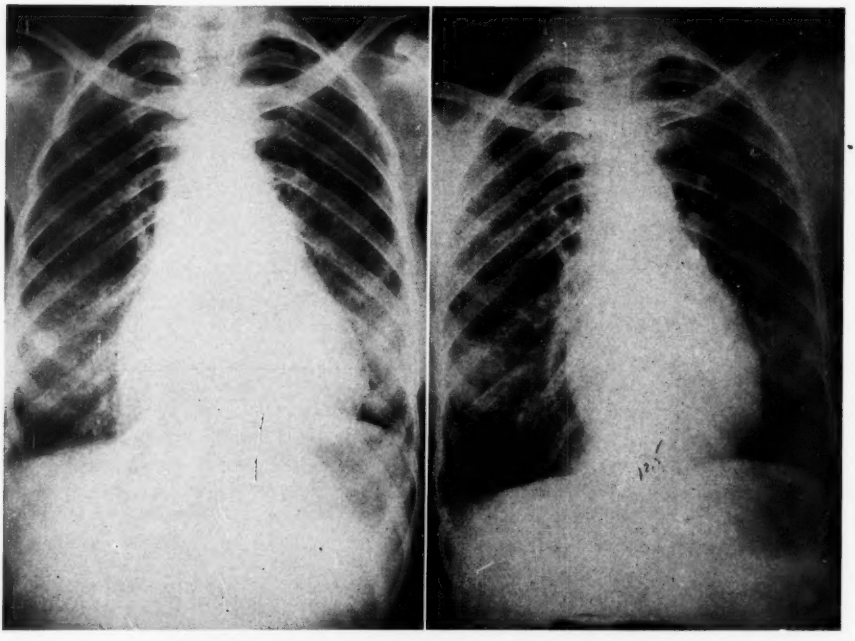


Fig. 4.—Case 1. Method of closure of aorticopulmonary fistula.



A. *B.*

Fig. 5.—Case 1. *A* and *B*, Preoperative and postoperative roentgenograms of the chest.

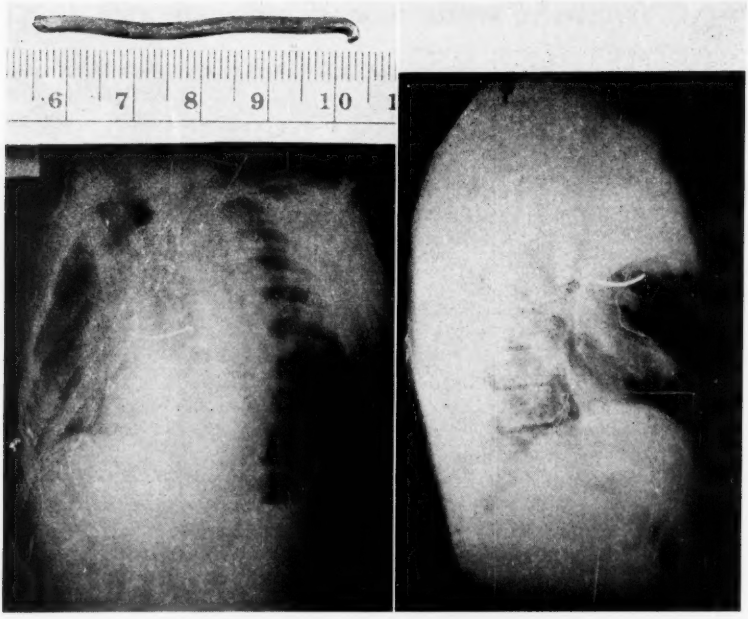
Examination of the chest again revealed a palpable thrill and a continuous harsh murmur in the second left intercostal space near the sternal border. Chest roentgenogram revealed enlargement of the cardiac shadow and some congestion of the lung fields. Fluoroscopy of the chest showed a slight hilar dance. A clinical diagnosis of traumatic aorticopulmonary fistula was made. Cardiac catheterization studies revealed the oxygen saturation in the right pulmonary artery to be not significantly higher than in the right ventricle. The left pulmonary artery could not be catheterized. A retrograde aortogram made through the left common carotid artery showed simultaneous filling of the aorta and the left pulmonary artery (Fig. 1).

Operation was carried out on June 10, 1952, utilizing bilateral anterior third intercostal space incisions with transverse section of the sternum. A thrill could be felt in the region of the proximal portion of the left main pulmonary artery on its superior aspect which could be obliterated with finger pressure. The aorta and left main pulmonary artery in this region were gradually exposed by dissection and a fistulous tract connecting the two vessels could then be identified (Fig. 2). After further dissection it was possible to apply suitable clamps to the aorta and pulmonary artery (Fig. 3). The fistulous tract was divided and was found to be 10 mm. in diameter. The aortic side of the fistula was first closed with a continuous lateral suture of 3-0 arterial silk brought back on itself as an over-and-over suture. The pulmonary artery side of the fistula was dealt with in a like manner (Fig. 4). There was no difficulty with bleeding when the clamps were removed. Following closure of the fistula, no thrill could be felt. Her postoperative course was uncomplicated. The postoperative blood pressure was about 120/80 mm. Hg. She was discharged from the hospital on the twentieth postoperative day.

The patient did well during the first few months following operation. There was a gradual decrease in the cardiac size (Fig. 5, *A* and *B*). The patient has subsequently been lost to follow-up.

Case 2.—E. S., a 28-year-old white man, experienced sudden sharp pain in the anterior chest while pushing a rotary lawn mower on June 22, 1958. He became dyspneic and fainted. He was seen by a physician who prescribed medication for pain. A roentgenogram of the chest was not made. During the next 24 hours he experienced anterior chest pain, dyspnea, and several syncopal attacks. He was seen by another physician about 36 hours after injury. A roentgenogram of the chest showed a piece of wire which was presumed to be in the mediastinum. He was hospitalized and the right chest was entered through a posterolateral incision. The mediastinum was explored and the foreign body was felt to be in the ascending aorta. There was no bleeding from the aorta at this time. The operative incision was closed and the patient was transferred to Vanderbilt University Hospital about 56 hours after injury.

The temperature was 101° F. on admission. He was moderately dyspneic and was uncomfortable from the recent right thoracotomy. Chest roentgenograms showed the right lung to be well expanded with some diffuse haziness at the right lung base. The presence of a foreign body in the mdiastinal region was confirmed (Fig. 6, *A* and *B*). Operation was undertaken soon after admission. He was placed on a cooling blanket and at the conclusion of the operation the temperature had fallen to a low of 87° F. The mediastinum was exposed by means of a median sternotomy incision. The ascending aorta was exposed and the right mediastinal pleura was opened. There was no collection of blood in the mediastinum; the anterior wall of the ascending aorta was edematous and there was a small blue area, 2 mm. in diameter, on the anterior wall of the aorta about 2 cm. above the aortic ring. The dissection was carried posteriorly and the foreign body could be felt protruding through the posterior aortic wall. The superior and inferior venae cavae were dissected free and suitable tapes placed about them; the distal ascending aorta was dissected free and a tape placed about it. Stay sutures were placed in the anterior wall of the aorta about 5 cm. apart. In-flow and out-flow stasis was then accomplished by occluding first the venae cavae and subsequently the aorta, distal to the stay sutures. An incision, 4 cm. long, was made through the anterior wall of the aorta. A piece of heavy rusty wire (Fig. 6, *A*) which measured almost 5 cm. in length,



A. *B.*

Fig. 6.—Case 2. *A* and *B*, Preoperative roentgenograms of the chest; the wire is shown in *(A)*.

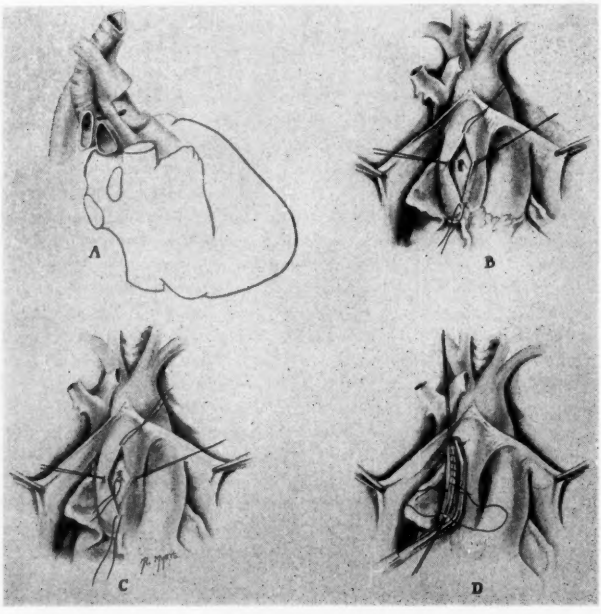


Fig. 7.—Case 2. Schematic drawing depicting the location of the wire and the method of its removal from the aorta.

approximately half of which protruded into the lumen through the posterior wall of the aorta, was removed with a hemostat, leaving a hole 3 to 4 mm. in diameter in the posterior wall of the aorta. This defect was closed with three interrupted 3-0 arterial silk sutures. A suitable curved clamp was then placed in such a manner as to occlude the defect in the anterior wall of the aorta after the venae cavae were released and the heart and ascending aorta allowed to fill with blood. The circulation had been interrupted for exactly 4 minutes. Following restoration of the circulation, the aortic wall was sutured with a continuous suture of silk after which the tangential occluding clamp was removed; however, there was brisk bleeding from the suture line and the clamp was re-applied and the suture line re-enforced with 4-0 arterial silk sutures after which there was no bleeding (Fig. 7). A drainage tube was left in the right pleural cavity, the sternum was approximated with four heavy wire sutures, and the remainder of the operative wound was closed with interrupted fine silk sutures.

The patient's course in the postoperative period was uneventful until the second postoperative day when his temperature rose to 104° F. A blood culture was obtained. He was at this time receiving 10 million units of aqueous penicillin intravenously twice daily. He developed signs of retention of secretions in the right lung and a tracheostomy was performed. He continued to have daily temperature elevations to 104° F. and blood cultures were obtained daily. There was no growth of organisms in the first three blood cultures. Because of the continued high fever, chloramphenicol (0.5 Gm. every 6 hours) and kanamycin (0.5 Gm. every 6 hours) were added to the treatment program. On this therapy the daily elevation of temperature gradually became less marked. A blood culture drawn on the thirteenth postoperative day revealed *Escherichia coli* bacillus which was sensitive to chloramphenicol and kanamycin. These drugs were continued and he became afebrile on the twenty-fourth postoperative day. He was discharged from the hospital on the twenty-eighth postoperative day. He has been well since leaving the hospital and has returned to work as a farmer.

CASE 3.—J. D. J., a 46-year-old Negro man, was stabbed in the left anterior second interspace near the sternal border on April 26, 1958. He was admitted to a hospital soon after the injury and was found to have evidence of blood in the left pleural cavity; otherwise, examination of the heart and lungs revealed no abnormality. He was given blood transfusions and penicillin. On May 9, 1958, a drainage tube was placed in the left pleural cavity and 1 L. of bloody fluid removed. On May 21, 1958, left thoracotomy was performed for the purpose of decortication but the pleural cavity contained only 800 c.c. of thin bloody fluid and decortication was not required. No other abnormality was noted at that time.

After recovering from this procedure he was discharged from the hospital. He developed progressive dyspnea, palpitation, fatigability, and ankle edema. On July 7, 1958, he was found to have a loud continuous murmur over the base of the heart and signs of cardiac failure. He was admitted to Thayer Veterans Administration Hospital at this time for further study.

Examination revealed a prominent thrill in the second intercostal space at the left sternal border and a continuous harsh murmur was heard over the entire precordium which was loudest at the left sternal border in the second and third interspaces. The cardiac rhythm was regular; blood pressure was 110/70 mm. Hg. The liver was enlarged to four fingerbreadths below the right costal margin and a moderate degree of ankle edema was present. Chest roentgenograms revealed slight enlargement of the cardiac shadow. Electrocardiograms were considered to be within normal limits. The clinical impression was of a probable aorticopulmonary fistula.

Cardiac catheterization studies revealed an increased cardiac output and increased pulmonary artery pressure. Oxygen saturation levels were 62 per cent in the superior vena cava, 68 per cent in the right atrium, 71 per cent in the right ventricle, and 71 per cent in the main pulmonary artery. A retrograde aortogram performed through the right brachial artery showed simultaneous filling of the aorta and the pulmonary artery (Fig. 8).

He was digitalized and was then transferred to the Vanderbilt University Hospital where operation was performed on Sept. 30, 1958. A bilateral anterior thoracotomy incision through the third intercostal space with transection of the sternum was employed. A few adhesions were encountered which involved the pericardium and pleura. A continuous thrill was felt in the pulmonary artery and a fistula between the base of the aorta and the pulmonary artery was easily identified. The fistula could be closed by finger pressure. The defect was located about 2 cm. above the pulmonary annulus. No scar was apparent on the surface of the aorta or pulmonary artery. Palpation over the wall of the right atrium revealed a vigorous jet of blood entering this chamber presumably from the base of the aorta, which could be abolished by finger pressure. A finger was inserted into the right atrium through the auricular appendage and a jet of blood was felt to come through a small cone-like projection in the atrial wall.

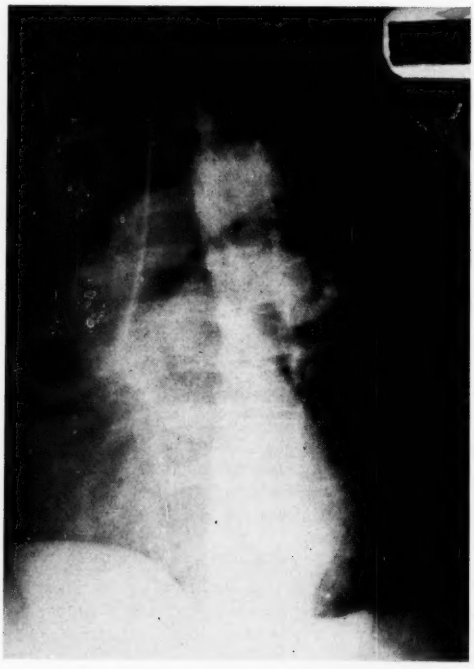


Fig. 8.—Case 3. Retrograde aortogram which shows simultaneous filling of the aorta and pulmonary artery.

Extracorporeal circulation was then established and cardiac arrest was induced with potassium citrate. A linear incision was made through the anterior wall of the ascending aorta; a slit-like fistula between the aorta and the pulmonary artery was seen. The opening was 12 to 14 mm. in length by 3 mm. in width. The defect was closed with interrupted sutures of 3-0 silk from within the aorta. The right atrium was then opened; a similar slit-like fistula was visualized which extended through the right side of the ascending aorta from a point just above the annulus in the region of the noncoronary sinus but not extending down into the sinus. The aortic entrance of this fistula was about 10 mm. in length and 2 to 3 mm. in width. Viewed from the atrial side, a redundant portion of atrial endothelium was found which was lifted to reveal a slit-like opening,

about 5 mm. in length, which represented the atrial side of the fistula. The aortic component of the aortico-atrial fistula was closed from within the aorta with a row of interrupted 3-0 silk sutures and the atrial side was closed with two interrupted 3-0 silk sutures (Fig. 9, *A* and *B*). The incisions in the right atrium and aorta were then closed and normal circulation was established without difficulty. The postoperative course was complicated by atrial flutter which was easily controlled with digitalis.

Cardiac catheterization studies carried out 4 weeks after operation revealed normal findings.

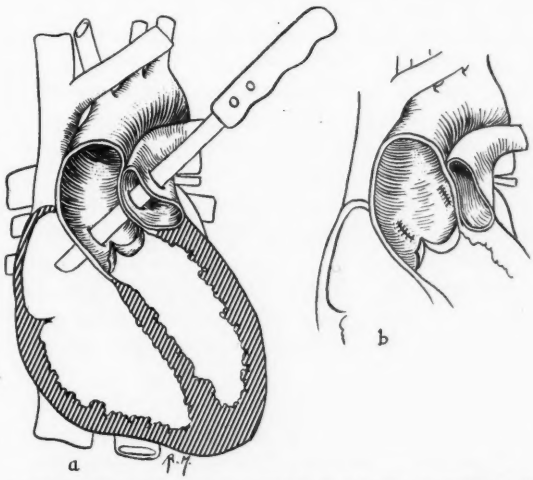
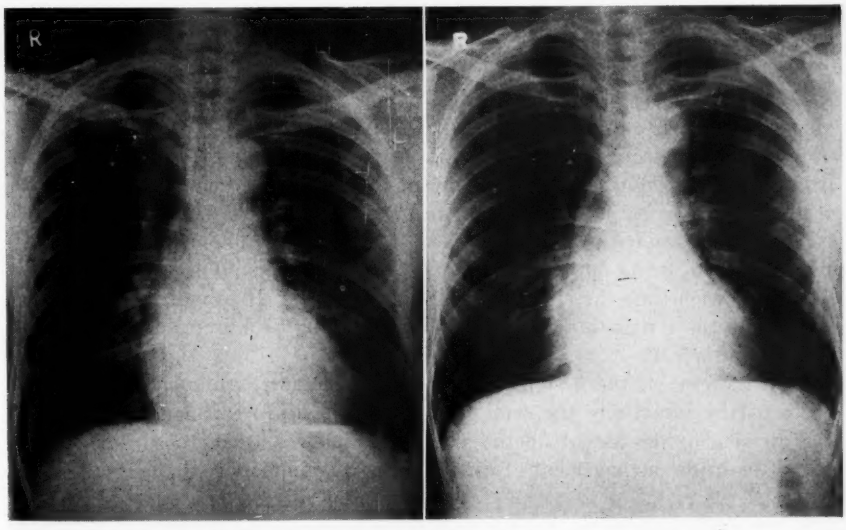


Fig. 9.—Case 3. *a* and *b*, Schematic drawing which depicts the position of the wounds of the pulmonary artery, aorta, and right atrium, and of the resulting fistulas.



A. *B.*

Fig. 10.—Case 3. *A* and *B*, Preoperative and postoperative roentgenograms of the chest.

He continued to do well after leaving the hospital; digitalis was discontinued 4 months after operation. He was last examined 9 months after operation at which time he was free of symptoms and no cardiac abnormality could be detected. Roentgenogram of the chest showed diminution in the size of the heart shadow when compared with the preoperative film (Fig. 10, *A* and *B*).

COMMENT

The fact that patients survive injuries such as are described here and elsewhere by authors, who have reported somewhat similar situations, is remarkable. Cases 1 and 3 received penetrating wounds which injured the walls of two great vessels which lay in proximity to each other—the aorta and pulmonary artery. Thus, a fistula was established between two vessels in which there is normally a high pressure differential, as occurs in the formation of arteriovenous fistulas. Both of these wounds were located within the pericardial sac. Cardiac tamponade occurred in the first patient. It seems plausible to reason that tamponade may have been lifesaving in this case in that the flow of blood from the aorta may have been slowed to the extent that thrombus formation in the aortic wound prevented further hemorrhage following pericardiocentesis. In the case of the third patient in this report, the penetrating knife continued its plunge to again perforate the opposite side of the aortic wall and the wall of the right atrium.

The wire, which traversed the ascending aorta of the patient in the second case report and which produced an extrapericardial wound, was bent in a barb-like manner at each of its ends. We believe it must have been moving at an extremely rapid speed to have caused such an injury without ensuing fatal hemorrhage. At the time of operation the tracheal wall could be palpated posterior to the aorta; it is believed that the wire extended through the anterior wall and into the lumen of the trachea.

The methods employed in the management of these lesions varied in each case. The presence of an intracardiac lesion in the third patient made the use of extracorporeal circulation necessary. Cardiopulmonary bypass was not employed in the second case because we were not prepared to use the pump-oxygenator at that time on very short notice. The presence of evidence of infection made us fear that secondary hemorrhage might be imminent. For this reason it was decided to proceed with operation at once under hypothermia, the plan being to use in-flow stasis for one or more periods of time, not to exceed 4 minutes each time the circulation was interrupted. It proved to be necessary to occlude the circulation for a total period of only 4 minutes and the patient tolerated this well.

In the case of the first patient the traumatic aorticopulmonary fistula was corrected by essentially the same techniques which are commonly employed in the division of the patent ductus arteriosus and which have been described by one of us in the management of congenital aorticopulmonary fistula.[13]

SUMMARY

The successful surgical treatment of 3 patients with wounds of the proximal aorta is described in detail.

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(For Discussion, see page 95)

ANTIBIOTICS AND EXTRACORPOREAL CIRCULATION

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FOLLOWING its demonstration by Rammelkamp and Maxton,[38] the awareness that certain bacterial organisms develop a resistance to commonly employed antimicrobials[28, 47] focused new attention on the problems of postoperative infection.[6, 7, 17-19, 22-26, 30, 46] The virulence of such resistant bacteria and the occasional near-epidemic frequency of nosocomial infections have been the subject of numerous reports.[9, 33, 42, 49] Initially it was hoped that antibiotic therapy given prophylactically might protect individuals against infections involving the operative wound, the pulmonary system, or the genitourinary tract in those circumstances in which these infections were not previously present. That prophylactic antibiotics do not prevent the occurrence of infections in comatose patients,[35] in patients after tracheostomy,[29] and after surgical procedures such as herniorrhaphy,[5] and various types of laparotomies,[32] but may actually favor the development of future resistant bacterial strains and a false sense of postoperative confidence, has been reported by many authors reviewing large groups of patients.[1, 21, 27, 40, 46]

Infection after thoracic and cardiovascular procedures has been discussed infrequently.[7, 16, 42] The occurrence of endocarditis after mitral and aortic valvotomy is verified by scattered case reports,[8, 12, 13, 20, 31, 34, 39, 43] although surveys of many patients undergoing cardiac surgery have been presented without noticeable infection.[3, 15] Infections in patients after operative procedures for congenital heart disease have been mentioned, particularly in relation to ductus arteriosus, and especially after its ligation, as well as after operations for atrial septal defect and pulmonic stenosis.[14, 41, 45]

The use of antibiotics in operations involving the colon, preoperatively and postoperatively, is generally agreed upon.[10, 11, 36, 37]

We should like to summarize the type and incidence of postoperative infections in all 222 patients who had mitral valvotomy, transection of ductus arteriosus, repair of coaretation of the aorta, and operations with the use of extracorporeal circulation at the University of Kansas Medical Center during the period from Sept. 15, 1957, to March 1, 1960. This is the period during which extracorporeal circulation has been used. The study was terminated on March 1 to include the first postoperative month. The patients have been divided into

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two main groups, those receiving antibiotics postoperatively as a prophylactic "umbrella" and those who did not receive prophylactic antibiotics during the postoperative period. In most instances those patients receiving antibiotics were observed during the first half of the period under survey, while those who did not were operated upon in the latter half of this time. We have not used prophylactic antibiotics routinely in patients undergoing cardiovascular procedures since March, 1959. With this exception, the groups are roughly similar. There has been no major difference in operating room techniques, operative management, or postoperative care.

We were interested in ascertaining whether or not any appreciable differences in postoperative infection were present between the group receiving antibiotics and the group that did not receive antibiotics prophylactically. "Prophylactic antibiotics" has been taken arbitrarily to include those antibiotics begun within 48 hours of operation, continued for several days without signs of infection being present, and in nontherapeutic doses.

Patients dying within the first 48 hours postoperatively were not considered since the period of observation was insufficient. Blood cultures were obtained from all patients at postmortem examination. No cultures were positive in those patients dying within the first 48 hours.

RESULTS

Coarctation of the Aorta.—The charts of 18 patients were reviewed. Seven received antibiotics postoperatively (5 penicillin and streptomycin, 2 penicillin, and, in 1 patient, administration of penicillin and streptomycin for 4 days was followed by tetracycline). Eleven received no prophylactic antibiotics postoperatively, although in 1 patient Dicrysticin was begun on the fourth postoperative day and continued until the seventh, the indication being that of fever. In two instances, Dacron grafts were inserted. None of these 18 patients had any objective signs of infection.

The average of the postoperative day, when the last temperature was recorded over 100° F. in those patients receiving prophylactic antibiotics, was 4.1, with an average dismissal time of 12.4 days, postoperatively. In those patients who did not receive prophylactic antibiotics postoperatively the average postoperative day with a temperature over 100° F. was 3.8 and the average day of dismissal from the hospital was 10.7.

These two groups show no essential differences. They do indicate that the patients who did not receive postoperative prophylactic penicillin and streptomycin fared as well as those who did.

Ductus Arteriosus.—Forty-one patients constitute this group. Twenty of these received prophylactic postoperative antibiotics of usual dosage (14 received penicillin and streptomycin; 5, penicillin; and 1, tetracycline). Twenty-one received no antibiotics postoperatively. In none of these 41 patients was there any evidence of infection. In those patients receiving antibiotics prophylactically, the average postoperative day with the last temperature over 100° F.

was 3.0; the average day of dismissal from the hospital was 8.8. In those patients who received no antibiotics, the average of the last postoperative day with a fever of 100° F. was 1.7 and the average day of dismissal from the hospital was 7.8.

In 1 patient given prophylactic antibiotics, tracheostomy was required because of laryngeal edema secondary to endotracheal intubation. In 6 patients, a minor pleural effusion occurred, one of these being chylous. These 6 patients were receiving prophylactic antibiotics. No appreciable differences can be noted between the postoperative courses of these two groups with ductus arteriosus.

Mitral Valvotomy.—Fifty-five patients comprise this group. Nine patients received one injection of 1,200,000 units of procaine penicillin during their postoperative period as prophylaxis against rheumatic fever. Forty-nine of the 55 were given daily prednisone (usually 2.5 mg. twice daily) to aid in prevention of the "postvalvotomy syndrome." Thirty of the 55 patients received prophylactic antibiotics (17 received penicillin and streptomycin; 7, penicillin; 3, erythromycin; 1 erythromycin and streptomycin; 1 tetracycline and erythromycin, and 1 tetracycline).

In the 25 patients who did not receive prophylactic antibiotics postoperatively, 5 were subsequently given antibiotics. These were administered because of a questionable pneumonia in the left lower base of the lung (1 patient), of pyuria (1 patient), and of a subcutaneous minor wound infection due to nonhemolytic coagulase-positive *Staphylococcus aureus* (1 patient). In 2 patients, no indication can be ascertained from the charts for instituting antibiotic therapy. In these 5 patients to whom chemotherapy was subsequently given, penicillin and streptomycin were give in 2, tetracycline in 1, Gantrisin in another, and Furadantin was given to the patient with the wound abscess.

Because of the variable nature of the cardiopulmonary abnormality shown in these patients, duration of fever and day of hospital dismissal have not been tabulated. Many of the patients were kept in the hospital following recovery from their operative procedure for attempted conversion from atrial fibrillation to a normal sinus rhythm. However, the occurrence of infection is not significantly different in these two groups. The one wound abscess that did develop in a patient not receiving prophylactic antibiotics was resistant to penicillin and Chloromycetin, moderately sensitive to erythromycin and Aureomycin, and trace sensitive to streptomycin, Terramycin, and tetracycline.

Extracorporeal Circulation.—The records of 108 patients representing a variety of cardiac defects were available and reviewed. The only patients excluded during this period were those not surviving long enough for evaluation. These were divided into two groups, those with (46 patients) and those without prophylactic antibiotics (62 patients). In most instances the patients operated upon during the past year have not received antibiotic prophylaxis and accordingly this represents a later group of patients as compared with those earlier in our series.

In the 62 patients (Tables I-III) who did not receive prophylactic antibiotics, there were 14 instances of infections. In 3 patients there was pyelonephritis as manifested by pyuria, with or without fever. In 6 patients there were wound abscesses of varying degrees: two were due to hemolytic *Staphylococcus aureus,* coagulase positive; four to hemolytic *Staphylococcus aureus,* coagulase negative; in one of the latter abscesses, beta hemolytic streptococcus was also cultured. In 2 patients there were clinical, radiologic, and laboratory signs of a pneumonitis, although no definite organism was cultured. One of these (MR) died on the seventeenth postoperative day primarily from protracted congestive heart failure and bronchopneumonia. The drain site of the intrapleural tube was infected with hemolytic *Staph. aureus,* coagulase positive, although several blood cultures were consistently negative. No histologic signs of suppurative or staphylococcal infection were found at autopsy except for the drain site.

TABLE I

PROCEDURE	TOTAL PATIENTS	NO "PROPHYLACTIC ANTIBIOTICS"			"PROPHYLACTIC ANTIBIOTICS"		
		NO. PATIENTS	NO INFECTION	INFECTION	NO. PATIENTS	NO INFECTION	INFECTION
Repair of coaretation of aorta	18	11	11	0	7	7	0
Transection of ductus arteriosus	41	21	21	0	20	20	0
Mitral valvotomy	55	25	21	4	30	30	0
Extracorporeal circulation	108	62	48	14*	46	36	10†
Total	222						

*Two deaths.
†Five deaths.

TABLE II. INCIDENCE OF INFECTION AMONG PATIENTS HAVING HAD EXTRACORPOREAL PERFUSION

108 Patients

14 Infections in 62 without "prophylactic antibiotics"
- 3—Pyelonephritis
- 6—Wound abscess
- 2—Pneumonitis
- 1—Delayed empyema requiring readmission
- 2—Septicemia (staphylococcus)

10 Infections in 46 with "prophylactic antibiotics"
- 1—Pyelonephritis and pneumonitis
- 1—Pneumonitis
- 3—Wound abscess
- 5—Septicemia (1 Pseudomonas, 4 staphylococcus)

One patient (PM) was dismissed from the hospital after an uneventful convalescence following repair of a trilogy of Fallot only to be readmitted 2 weeks later with a left pleural effusion and congestive heart failure. Culture of the pleural fluid revealed several colonies of hemolytic *Staph. aureus,* coagulase positive. She responded to penicillin (600,000 units twice daily for 7 days) and appropriate measures for the treatment of her decompensation. Two patients (JR and JM) developed a generalized septicemia with positive blood cultures. One of these patients (JM) died on the eleventh postoperative day

TABLE III. INFECTIONS IN PATIENTS RECEIVING "PROPHYLACTIC ANTIBIOTICS" (62)

PATIENT	AGE	PROCEDURE	INFECTION	ORGANISM	ANTIBIOTICS*	COMMENT	DAY OF FEVER OVER 100° F.	DAY HOME
OK	43	Aortic valvotomy	Small wound abscess	*Staph. aureus*, hemolytic, coagulase +; *Staph. aureus*, nonhemolytic	None given	Treated by incision and irrigation only	4	15
DL	6	Ostium secundum closure	Small wound abscess	*Staph. aureus*, hemolytic, coagulase neg.	None given	Treated by incision and irrigation only	9	11
NM	6	Closure ASD	Small wound abscess	*Staph. aureus*, hemolytic, coagulase neg.	None given	Treated by incision and irrigation only	9	10
LG	37	Closure ASD	Small wound abscess	*Staph. aureus*, hemolytic, coagulase neg.	None given	Treated by incision and irrigation only	17	19
PM	13	Repair trilogy of Fallot	Fever weeks after dismissal with rt. pleural effusion and decompensation	*Staph. aureus*, hemolytic, coagulase +	Treated with penicillin on readmission	Recovered uneventfully after compensating congestive failure and penicillin	8	9
FS	39	Closure ASD	Small wound abscess	*Staph. aureus*, hemolytic, coagulase +	Treated with erythromycin; had been given tetracycline (4-9) for undetermined fever	Kept in hospital for conversion to normal sinus rhythm	17	30
MR	47	Repair mitral regurgitation	Drain site infection and pneumonitis	*Staph. aureus*, hemolytic, coagulase +	Penicillin and tetracycline (1); Chloromycetin (4-12); Ilosone (12-17)	Died 17th day with severe bronchopneumonia; 3 blood cultures negative. Had small stitch abscess from femoral embolectomy preoperatively (hemolytic *Staph. aureus*, coag. +, 80)		

EL	25	Repair of tetralogy of Fallot	Pneumonitis	--------	Chloromycetin (13-20) (25-34) (36-38)	Developed decompensation	38	39
SH	10	Pulmonic valvotomy	Pyelonephritis	--------	Gantrisin (7-9)	Uneventful recovery	7	10
PV	13	Repair of trilogy of Fallot	Small wound abscess	*Staph. aureus*, hemolytic, coagulase neg. and beta hemolytic streptococcus	Chloromycetin (12-17)	Tracheostomy required 2nd postop. day for copious secretions	9	17
MO	47	Aortic valvotomy	Pyelonephritis	--------	Furadantin (9-10)	Uneventful recovery	0	10
JR	12	Aortic valvotomy	Septicemia and wound abscess	*Staph. aureus*, hemolytic, coagulase +	Chloromycetin (4-11) Declomycin (10-13), Erythromycin (12-36)	Recovered	42	46
JM	13	Repair A-V communis	Septicemia	*Staph. aureus*, hemolytic, coagulase +	Gantrisin (5-7), penicillin (5-11), vancomycin (5-11)	Died 11th day postoperatively with overwhelming pneumonia, endocarditis, and pericarditis	11	11
JA	9	Repair of tetralogy of Fallot	Pyelonephritis	--------	Gantrisin (4-9)	Uneventful recovery	8	10

*Numbers in parentheses are days postoperatively that antibiotics were given.

TABLE IV. INFECTIONS WITHOUT "PROPHYLACTIC ANTIBIOTICS"*

PATIENT	INFECTION	ORGANISM	PHAGE	SENSITIVITY†						
				PENICILLIN	STREPTOMYCIN	AUREOMYCIN	TETRACYCLINE	TERRAMYCIN	CHLOROMYCETIN	ERYTHROMYCIN
OK	Wound	*Staph. aureus*‡	7, 29, 52, 79, 81, 83	M	M	M	M	--	M	M
DL	Wound	*Staph. aureus*, hemolytic, coagulase neg.	--------	M	M	M	R	T	M	M
LG	Wound	*Staph. aureus*, hemolytic, coagulase neg.	--------							
PM	Pleural fluid	*Staph. aureus*	--------	T	R	R	R	R	T	R
MR	Pneumonitis and wound	*Staph. aureus*	80	R	R	R	T	R	M	M
FS	Wound	*Staph. aureus*	29, 47	R	T	M	M	S	M	M
NM	Wound	*Staph. aureus*, hemolytic, coagulase neg.	--------	T	M	R	R	R	M	M
MV	Wound	*Staph. aureus*, hemolytic, coagulase neg.	--------	R	T	M	T	T	R	M
PV	Wound	Beta hemolytic streptococcus	--------	M	M				M	M
JR	Septicemia and wound	*Staph. aureus*	29, 44A, 52, 75, 79, 81	T	T	M	S	M	M	S
JM	Septicemia and wound	*Staph. aureus*	29, 52, 81	T	T	M	M	M	M	M

*Only those infections are listed with cultured organisms and tested sensitivities.
†R = resistant; M = moderately sensitive; T = trace sensitive; S = sensitive.
‡*Staphylococcus aureus*, coagulase positive, hemolytic, unless otherwise specified.

with an overwhelming staphylococcus infection as manifested by a pyopericarditis, bronchopneumonia, and severe endocarditis, despite intensive treatment with vancomycin and penicillin. The other patient (JR) who also had a minor staphylococcal wound abscess recovered satisfactorily after a month's therapy with vancomycin. Thus, in the group of 14 infections there were two deaths, one of which can be attributed directly to overwhelming staphylococcal septicemia.

The organisms cultured, their phage type, and sensitivities are shown in Table IV.

In the 46 patients who were given prophylactic antibiotics: 27 received penicillin and streptomycin, of whom 2 were changed to Chloromycetin and another to erythromycin with the later addition of Chloromycetin; 3 received penicillin; 11 received tetracycline, 2 later being changed to Chloromycetin and 1 to penicillin; 3 received Chloromycetin with 1 having penicillin added and another being changed to Ilosone; 1 was given erythromycin and Chloromycetin; and 1 Aureomycin. Ten infections developed in this group (Table V). One patient (BN) had a questionable pneumonitis and a definite pyelonephritis. One had the clinical features of a left lower lobe pneumonitis (FJ), 3 had wound abscesses, two of which were due to hemolytic *Staph. aureus,* coagulase positive (MP and CP), and 1 in which no organism was cultured (EH). Five patients (EK, MT, WI, CP, JW) developed septicemia. One (WI) had severe rheumatic aortic insufficiency. The valve leaflets were sutured together to correct the insufficiency. On the third postoperative day, disruption of the valve leaflets occurred as noted by the sudden onset of a diastolic murmur and a widening of the pulse pressure. Concomitantly he developed a high fever and subsequently died on the fifth postoperative day. Blood cultures revealed *Pseudomonas aeruginosa*—no sensitivity studies were done. The other 4 patients developed septicemia due to hemolytic *Staph. aureus,* coagulase positive. These patients died on the fifth, seventh, eleventh, and forty-ninth postoperative days. The phage type and sensitivities of the organisms in this group are listed in Table VI.

MT had numerous vascular emboli at the time of mitral valve repair from a large clot in the left atrium that partially occluded several of the pulmonary vein orifices. Postoperatively the patient was febrile, comatose, and died on the seventh postoperative day. CT, with a ruptured sinus of Valsalva, pursued a febrile course postoperatively until death on the forty-ninth day. JW developed a transfusion reaction with anuria, postoperatively. An attempt to relieve the hyperkalemia was made by perfusion with an artificial kidney. Several days after this procedure the patient showed a blood culture positive for hemolytic *Staph. aureus,* coagulase positive, organisms. EK, operated upon for a trilogy of Fallot, pursued a febrile course until death on the fourth postoperative day. Postoperatively, her course was characterized by hypotension. Blood cultures taken at autopsy revealed hemolytic *Staph. aureus,* coagulase positive. Her death is believed primarily to be due to cardiovascular conditions complicated by septicemia. From this group of 10 patients with infections, five deaths occurred—all were due to septicemia.

TABLE V. INFECTION IN PATIENTS RECEIVING "PROPHYLACTIC ANTIBIOTICS" (46)

PATIENT	AGE	PROCEDURE	INFECTION	ORGANISM	ANTIBIOTICS GIVEN*	REMARKS	DAY FEVER OVER 100° F.	DAY HOME
FJ	8	Closure VSD	Pneumonia	--------	Penicillin and streptomycin (1-6), Chloromycetin (6-16), Mycostatin (16-20)	Recovered	18	20
MP	6	Closure VSD	Small wound abscess	*Staph. aureus*, hemolytic, coagulase +	Tetracycline (2-5), Chloromycetin, and penicillin (10-17)	Small localized abscess; (?) pneumonitis postoperatively; recovered uneventfully	14	17
EK	16	Repair tetralogy of Fallot	Septicemia	*Staph. aureus*, hemolytic, coagulase +	Tetracycline for 5 days	Died 5th day postop.; cardiac death stressed by septicemia, culture obtained at autopsy	5	--
CP	44	Repair mitral regurgitation	Major wound abscess	*Staph. aureus*, hemolytic, coagulase +	Tetracycline for 18 days	Wound abscess with sternal disruption at transverse thoracotomy; died on readmission one month later in decompensation	18	19
EH	13	Repair inf. stenosis	Small wound abscess	None	Penicillin (1-4), Chloromycetin (5-9), penicillin (11-13), Furadantin (16-19)	Dismissed on 29th postop. day; fever to 29th postop. day—cause undetermined; recovered satisfactorily		

MT	41	Repair mitral regurgitation	Septicemia	*Staph. aureus*, hemolytic, coagulase +	Tetracycline (1-4), Chloromycetin (5-7)	Had multiple cerebral emboli; cardiac death complicated by septicemia; died 7th postop. day	7	--
WI	24	Repair of aortic insufficiency	Septicemia	*Pseudomonas aeruginosa*	Tetracycline (1-5), penicillin (5-7)	Died 7th day; sutures approx. valve leaflets had torn loose, were the site of vegetations; septicemia aggravated by severe aortic insufficiency	7	--
CP	15	Repair ruptured sinus Valsalva	Septicemia	*Staph. aureus*, hemolytic, coagulase +	Chloromycetin (2-10), Ilosone (10-20), vancomycin (8-38)	Died 49th day postoperatively; vegetations on aortic cusp	18	20
JW	27	Repair mitral regurgitation	Septicemia	*Staph. aureus*, hemolytic, coagulase +	Chloromycetin (1-11), penicillin (10)	Transfusion reaction with oliguria—on artificial kidney. Died 11th postop. day; no infection noted postop. other than pos. blood culture	11	11
BN	15	Closure ASD	Pyelonephritis and (?) pneumonitis	--------	Penicillin and streptomycin (1-8), Furadantin (10-11), Chloromycetin (13-16)		13	17

*Numbers in parentheses are days postoperatively that antibiotics were given.

TABLE VI. SENSITIVITIES: INFECTIONS WITH "PROPHYLACTIC ANTIBIOTICS"

PATIENT	INFECTION	ORGANISM	PHAGE	SENSITIVITY*							ANTIBIOTICS USED PROPHYLACTICALLY
				PENICILLIN	STREPTOMYCIN	AUREOMYCIN	TETRACYCLINE	TERRAMYCIN	CHLOROMYCETIN	ERYTHROMYCIN	
MP	Wound	*Staph. aureus*†	7	M	M	M	M	M	M	M	Tetracycline (2-5), penicillin and Chloromycetin (10-17)
CP	Wound	*Staph. aureus*	77	R	R	R	R	R	T	M	Tetracycline (1-18)
MT	Septicemia	*Staph. aureus*	80	R	R	R	R	R	M	M	Tetracycline (1-9), Chloromycetin (5-7)
CP	Septicemia	*Staph. aureus*	80, 81	R	M	M	M	M	M	M	Chloromycetin (2-5), Ilosone (10-20)
JW	Septicemia	*Staph. aureus*	6, 7, 42E 47, 54, 77, 81	R	R	M	M	M	R	M	Chloromycetin (1-11), penicillin (10)
EK	Septicemia	*Staph. aureus*	NT‡	R	R	R	R	R	M	M	Tetracycline (1-5)

*R = resistant; M = moderately sensitive; T = trace sensitive.
†*Staphylococcus aureus*, coagulase positive, hemolytic.
‡Non-typable.

DISCUSSION

Our data (Table I) and the experiences of others with extracorporeal perfusion indicate that the incidence and gravity of infections is greater in patients having this type of procedure than with other types of cardiovascular operations. Undoubtedly many factors are responsible for this, no one of which applies to every situation or no one of which can be indicated for each infection or series of infection. In our patients, the comparative lack of infection in patients with cardiovascular operations in which the pump-oxygenator was not used, emphasizes the difference from those in whom extracorporeal perfusion was done. The same sterilization techniques, essentially the same personnel, the same operating room area, etc., make these two groups basically comparable since the groups were concomitant. Additional sources of infection exist because of the additional personnel in the operating room when extracorporeal perfusion is used and also from the assembly and priming of the apparatus. Another feature contributing to the greater likelihood of infection in these patients is the presence of anatomically deformed valves or abnormal defects that may act as a nidus for infection. The occurrence of endocarditis in these patients is a frequent and well-recognized complication of their disease under essentially normal circumstances. The importance of suture material or prosthetic devices in the area of surgical correction has been emphasized as a focus engendering bacterial colonization.[2, 44] It is indicative that in our 7 patients in whom septicemia developed, the anatomic abnormality in all involved a damaged valve (three mitral, three aortic, one pulmonic). In our group of patients having repair of atrial and ventricular septal defects numerically larger than those having valvular repairs, there has been no instance of postoperative septicemia.

The results in our 222 patients undergoing operation do not indicate fewer infections in those who received prophylactic antibiotics (penicillin, streptomycin, Aureomycin, tetracycline, etc., either singly or in combinations) than in those who did not receive prophylactic antibiotics. Our series does not permit statistically significant conclusions, but more deaths and more serious infections occurred in those patients who initially received prophylactic antibiotics than in the opposite group (Tables II, III, V). In addition, organism sensitivity was greater, that is, resistance was less, when antibiotics had not been given prophylactically (Tables IV and VI).

Some physicians have kept pace with the development of antibiotic restraint strains by changing the antibiotic for prophylaxis as resistance is acquired or by giving large, almost therapeutic, doses of antibiotics routinely in the postoperative period.[16] The routine administration of therapeutic doses of antibiotics does not seem a logical solution to the problem of postoperative infections for obvious reasons.

That antibiotics given prophylactically do not reduce postoperative infections has been noted by others[1, 5, 21, 24, 25, 32, 40] in a variety of "clean" surgical operations. Whether the use of postoperative prophylactic antibiotics is an innocuous or an insidious practice brings into consideration the development

of resistant strains by several types of bacteria in an antibiotic environment. It is well recognized that, in the presence of antibiotics, resistant strains of staphylococci, proteus, and pseudomonas are more prevalent than in the absence of antibiotics. To prophylactic antibiotics has been attributed an increasing number of resistant strains of bacteria, thus increasing the virulence of infections and intensifying the difficulty of therapy. In addition, the false sense of security generally imparted by prophylactic antibiotics may detract from necessary and proper urgency in detecting infections and instituting appropriate therapy based on sensitivity studies.

SUMMARY

1. Postoperative infection in 222 consecutive patients having mitral valvotomy, transection of a ductus arteriosus, repair of coarctation of the aorta, and operation with extracorporeal perfusion is reviewed. The patients have been subdivided into those who received prophylactic antibiotics and those who did not.

2. The occurrence of infection was as great in those patients receiving prophylactic antibiotics as in those who did not. Generally, organism sensitivity in the former group was less.

3. Our experiences do not support the policy of routine antibiotic prophylaxis postoperatively for patients having thoracic cardiovascular procedures. The possible disadvantages of prophylactic antibiotics are discussed.

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(There was no discussion of this paper.)

ACHALASIA OF THE ESOPHAGUS IN CHILDREN

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ACHALASIA or megaesophagus is a disorder of the emptying mechanism of the lower esophageal segment which is apparently due to a failure of sphincter relaxation. It appears as a functional obstruction of the cardia associated with marked dilatation and hypertrophy of the proximal esophageal wall (Fig. 1). The disease is well recognized in adults, although largely overlooked as a clinical entity in children. Achalasia is rare in children, constituting 4 per cent of the cases in all age groups. The presenting features are often confusing; nonetheless, successful treatment can be so rewarding that the condition deserves careful attention.

CLINICAL FEATURES

The onset of achalasia is insidious and is often difficult to determine accurately in children. None of our patients was under 1 year of age. The characteristic symptoms were present in 1 patient for 4 years before the correct diagnosis was made. Weight loss, dysphagia, regurgitation and aspiration with pulmonary complications dominate the clinical histories. In contrast to achalasia in adults, pain is not a common complaint. Initially there is a slight delay in the passage of foods in the esophagus into the stomach. This first appears intermittently but, as the disease progresses, the dysphagia becomes persistent. Since achalasia may develop early in life, a child may accept the disordered swallowing mechanism without appreciating that it is abnormal for, unlike the development of achalasia in the adult, the child may never have known a normal period that would make his dysphagia obvious. Many children acquire habits to overcome the difficulties such as gulping fluid, breathing deeply, and crouching forward while eating.[1, 2] With this in mind, it is easy to understand that although dysphagia is a common presenting complaint, it has usually been present for a long period of time before an accurate diagnosis is made. As the disease progresses, regurgitation develops and may be first noted as food remnants on the child's sleeping pillow. Five of our 6 patients gave such a history of nocturnal regurgitation. Associated with the regurgitation may be nocturnal coughing. This latter feature was present in 3 of our 6 cases, although overlooked before the

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diagnosis of achalasia was made on other grounds. Such episodes explain the relative frequency of pulmonary complications due to aspiration. Bronchitis, pneumonitis, bronchial obstruction with atelectasis, bronchiectasis, and empyema have been reported. One of our patients was diagnosed as having bronchitis on several occasions and received treatment for a long time before the true nature of the disease was discovered.

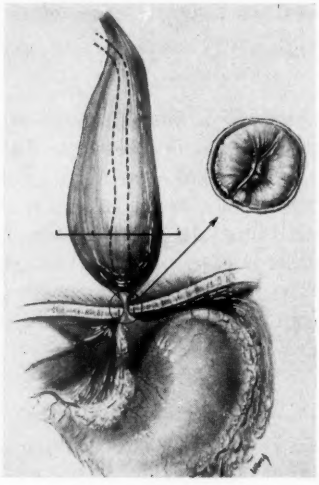

Fig. 1.—Semischematic drawing of achalasia which shows its relationship to the normal esophagus.

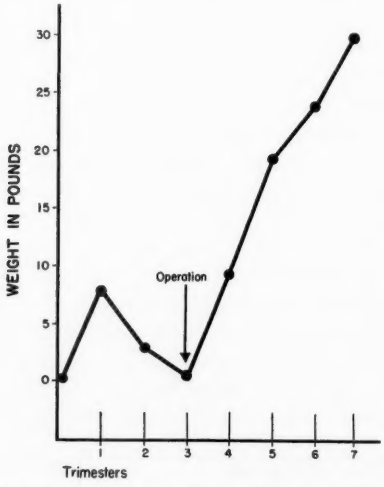

Fig. 2.—Diagram which shows weight gain postoperatively.

Weight loss was the outstanding feature in all 6 cases. The loss appeared proportionate to the degree of physiologic obstruction. Usually a child is unable to finish a meal and then becomes hungry 1 or 2 hours after the meal and seeks snacks before the next meal. One child had loss of weight over a 2-year period and, when seen at the age of 12, weighed only 58 pounds; after operation the patient gained 30 pounds (Fig. 2). Substernal pain, so often experienced by adults with achalasia, is a much less common complaint in children. In addition to the more important features, the literature contains reports of hoarseness of the voice, due to pressure on the recurrent laryngeal nerve, and cardiovascular disturbances, such as bradycardia. Such complaints were not present in our patients.

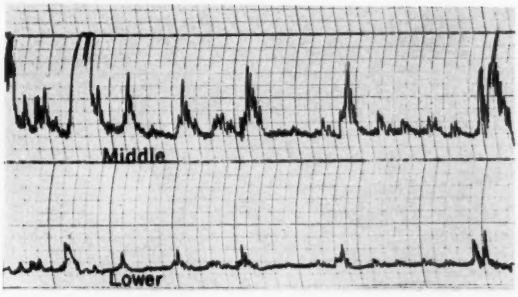

Fig. 3.—Peristaltic tracing from middle third and lower third of esophagus. Amplitude of contractions reduced in lower esophagus.

ROENTGENOGRAPHIC FINDINGS

The diagnosis of achalasia in children is based on the clinical history and roentgenographic studies. Occasionally the diagnosis can be established on a chest film showing widening of the mediastinum. Two of our cases were correctly diagnosed on a simple chest roentgenogram. On the barium swallow, there is smooth narrowing at the cardia with dilatation which is greatest in the lower half. Depending upon the severity and duration of the disease, the entire esophagus becomes dilated and tortuous. Occasionally esophagoscopy is of value in differentiating achalasia from stenosis due to peptic esophagitis or chemical burns, although, generally, achalasia creates less of an acute problem and does not have a comparable clinical history.

PATHOLOGY

In 1914 Hurst[3] suggested congenital absence or a degeneration of the ganglion cells in Auerbach's plexus as the pathologic disorder. Rake[4] in 1927 demonstrated degeneration of ganglion cells in 2 adult patients with achalasia. It is now established that patients with acquired megacolon have a degenerative lesion of Auerbach's ganglion cells. About 20 per cent of these patients have achalasia of the esophagus. In Hirschsprung's disease there is a congenital

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absence of Auerbach's ganglion cells in the distal colon. In 200 cases of aganglionic megacolon, we have not detected any esophageal lesion. In the 2 patients whom we operated upon, a strip of tissue was removed from the distal esophagus and no ganglion cells were found. Several similar strips of normal tissue removed at postmortem contained ganglion cells. Peristaltic studies on these 2 patients revealed active contractions. In 1, the amplitude of the contractions was normal (Fig. 3). The second patient had some diminution of amplitude. Experimental denervation of the dog's lower esophagus has produced cardiospasm and esophageal dilatation.[5] These findings suggest that the pathologic change in this disease is a parasympathetic defect in the lower esophagus. In children this is probably of congenital origin.

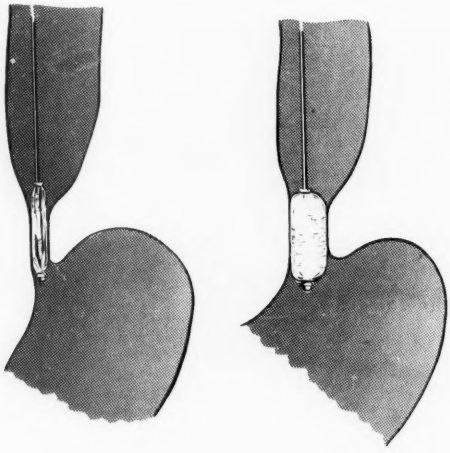


Fig. 4.—*A*, Diagram of a plummer pneumatic dilator in place. *B*, Diagram of dilator with bag inflated to a pressure of 8 pounds per square meter.

TREATMENT

Diet plays an unimportant role in the treatment of achalasia.[6] The anticholinergic drugs similarly are of little value. No improvements of esophageal function can be shown following the use of such agents and, in some cases, the condition was aggravated.[7] All of our patients were placed on anticholinergic drugs during the early part of our experience without success. The most satisfactory results are obtained with instrumental dilatation of the esophagus[1, 8] and this treatment was performed on all our patients. The dilatation can best be performed by a properly applied Plummer pneumatic dilator.[9] The bag dilator was inflated to a pressure of 8 pounds per square meter, being maintained in position for 5 minutes (Fig. 4). Following the first dilatation, the patient was discharged and nearly all children achieved immediate improvement. However, over the course of several months most of the children had recurrent symptoms and dilatation was repeated 6 months after the first treatment. By this regime, 4 of our 6 patients were satisfactorily managed

with a complete relief of symptoms and weight gain. Several other studies reveal that 70 to 80 per cent of the patients so treated have satisfactory results. Raia and Campos,[10] in Brazil, studied 140 cases of acquired achalasia in which there was treatment by dilatation of the cardia. They came to the conclusion that dilatation is the preferred procedure for the treatment of this condition as it is the most benign, the easiest, and least costly procedure. The complications of esophageal dilatations, such as rupture, aspiration, pneumonia, hematemesis, are rare in experienced hands.

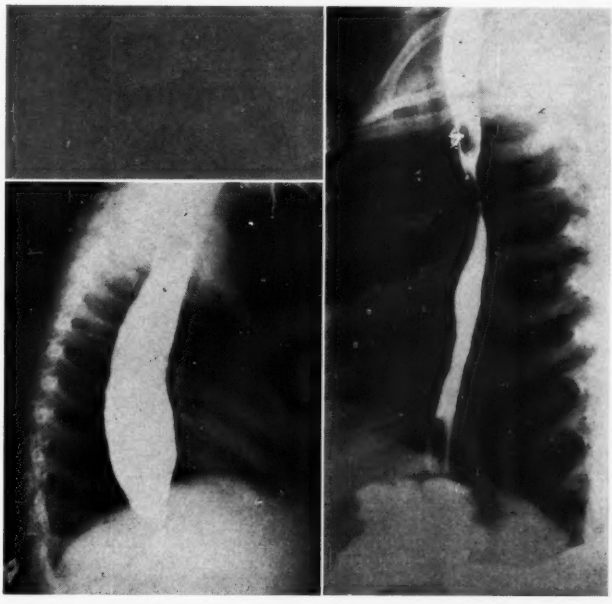


Fig. 5. Fig. 6.

Fig. 5.—Roentgenogram of achalasia when diagnosis was first made in Case 2.
Fig. 6.—In Case 2 the esophagus appeared normal 3 years after dilatation.

CASE REPORTS

CASE 1.—P. McE., an 11-year-old white boy, was admitted because of dysphagia and vomiting that had been present since infancy. At 3 months of age he had frequent episodes of dysphagia with associated vomiting. At 16 months of age, the dysphagia became resistant and nocturnal staining of the pillow by food particles with nocturnal cough was noted. His diet was limited to liquids and soft food and during the months prior to admission he had lost approximately 20 pounds in weight. At this time the child appeared well developed but greatly undernourished and chronically ill. Examination was otherwise not remarkable. Chest roentgenogram was normal. Barium swallow revealed moderate dilatation of the esophagus with smooth constriction at the cardia. The patient had two esophageal dilatations and his complaints were entirely relieved.

CASE 2.—C. B., a 3-year-old white girl, was admitted with a chief complaint of regurgitation of food since she was 8 months of age. During the months prior to admission she vomited undigested food and had failed to gain weight. She had no other complaints. On

examination she was underweight. Roentgenographic examination with barium revealed definite narrowing at the lower end of the eosphagus with marked dilatation of the proximal esophagus (Fig. 5). On esophagoscopy there was marked resistance in the cardia. After the first dilatation she had recurrent regurgitation which required a second treatment. Following this, she was active with normal growth and development. Four years later she had mild substernal pain after meals. She was again treated by dilatation and has remained free of symptoms for 3 years (Fig. 6).

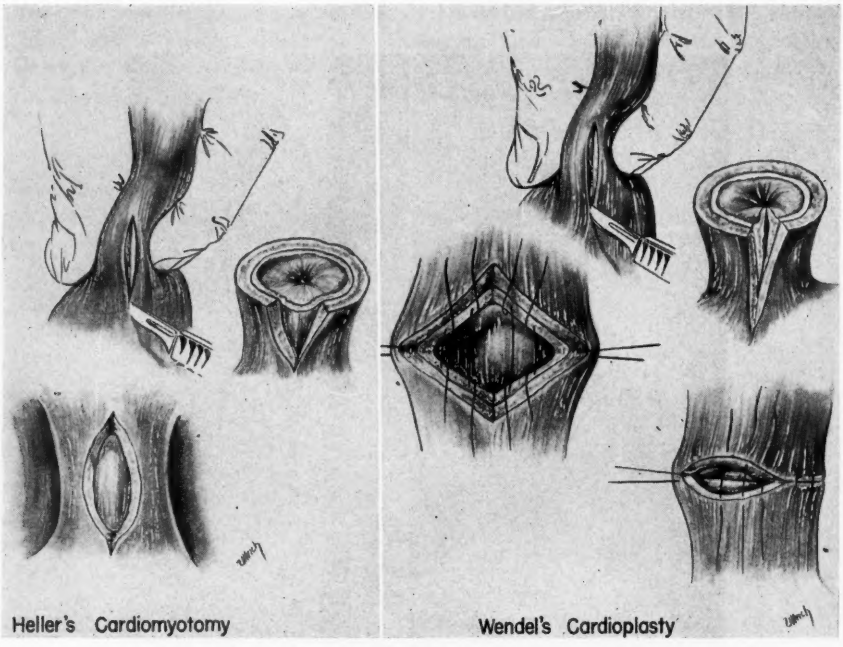


Fig. 7.—Semischematic drawing of Heller's and Wendel's operative techniques for achalasia. In Heller's operation the muscular coat is incised and the mucosa remains intact. In Wendel's procedure the mucosa as well as the muscular coat is opened and the incision closed in the opposite plane.

Surgical intervention is the treatment of choice in the approximate 20 per cent of the children who do not respond satisfactorily to dilatation. An ideal surgical procedure for achalasia in children has not yet been devised. At the moment, Heller's operation seems to be the best for the mild and moderate degree of achalasia and the Wendel operation for the advanced cases.

Gammelgaard[11] reports 84 per cent good results with the Heller's cardiomyotomy. The operation consists of a 1 to 2 cm. myotomy extending above and below the stenosis over the anterior wall of the distal esophagus, exposing the mucosa[12] (Fig. 7). Either the abdominal or the transthoracic routes can be used. However, the thoracic approach through a left intercostal incision was used in our 2 cases and provides an easier access to the lower esophagus than the abdominal route.

The Wendel operation[13] has been popularized in recent years by Sweet.[14] A vertical incision through all layers is made in the distal esophagus and cardia, which is then sutured transversely to enlarge the opening (Fig. 7). Many excellent results have been reported[15]; however, it and the Heller procedure are open to the objection that the acid gastric contents may regurgitate into the esophagus predisposing the peptic esophagitis. In 2 of our patients, no evidence of peptic esophagitis was noted.

Primary resection, which was described by Wangensteen[16] in 1951, is not recommended in children because it is too radical a procedure considering the benign nature of the underlying disease and the satisfactory results obtained by the simpler procedure that have been described.

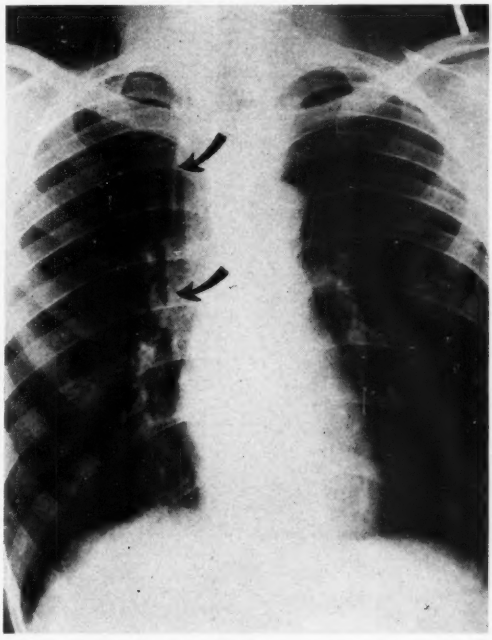
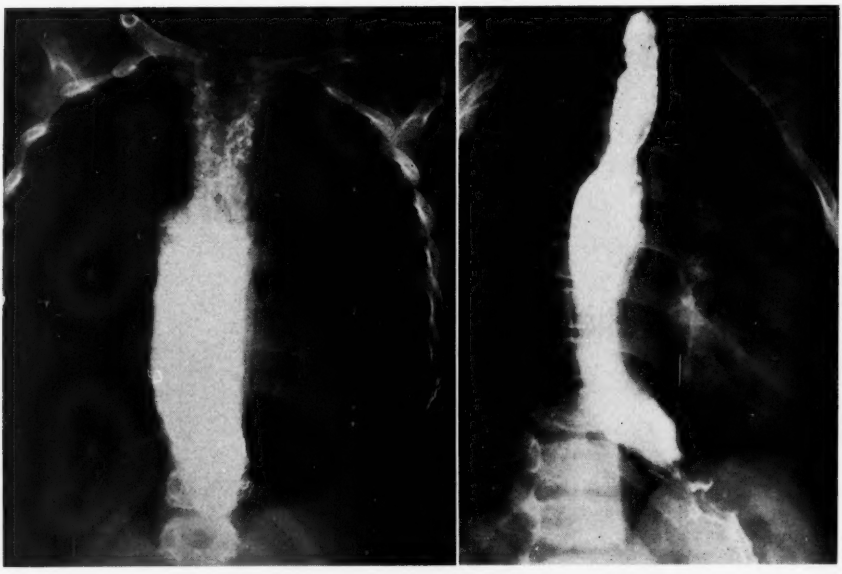


Fig. 8.—Chest roentgenogram with arrows indicating the curved linear shadow produced by the enlarged esophagus.

CASE 3.—J. B., a 12-year-old white boy, was admitted with a 4-year history of nocturnal cough, nocturnal vomiting, and recurrent upper respiratory infections. A year prior to admission he experienced mild dysphagia, became somewhat anorexic, and lost weight. In addition, he developed occasional gasping respirations and assumed peculiar positions in bed. His illness had been a constant source of anxiety to the boy and his parents. On previous admissions, diagnosis of acute pansinusitis and chronic bronchitis was made. On examination he was poorly nourished. A chest roentgenogram revealed a curved linear density paralleling the right lateral margin of the mediastinum, and lung field changes consistent with chronic infection (Fig. 8). Contrast media outlined a dilated esophagus ending in a narrow cardia (Fig. 9, *A*). During a year he was treated with three esophageal dilatations. Each was followed by temporary relief. At operation, the cardia was narrow but no evidence of

anatomical stricture was seen. Biopsy of the cardioesophageal function contained no ganglion cells. A Wendel cardioplasty was performed; postoperative course was uneventful. In the subsequent year the patient gained 30 pounds (Fig. 9, *B*). Four years postoperatively, he remains symptom free.

CASE 4.—J. P., a 2½-year-old white girl, was admitted with a 7 month's history of frequent regurgitation of a large amount of yellow white mucus, with an associated weight loss of 9 pounds and with minimal persistent cough. She was well developed and poorly nourished. Chest roentgenogram revealed a widening of the mediastinum to the right. A barium swallow was obtained and a dilated esophagus with a narrowing of the cardia and persistent delay in passage of the barium was demonstrated (Fig. 10). Dilatation on six occasions, including a manual dilatation through a gastrostomy, failed to give prolonged



A. *B.*

Fig. 9.—*A*, The barium swallow when the diagnosis was made. *B*, Eighteen months after surgery. Barium passes through the cardia without delay although the esophagus remained moderately dilated.

relief. A transthoracic cardioplasty was performed when she was 3½ years of age. In the 2 years after operation, she gained 13 pounds. Recent cinefluoroscopic studies with barium were reported as normal (Fig. 11) with fair peristaltic activity and without evidence of reflux. She remains asymptomatic 4 years after surgery.

COMMENTS

Our results obtained in these 6 cases are summarized on Table I. This condition may occur in children of any age. In some cases the symptoms began during early infancy. Grob and co-workers[17, 18] stated that there is no peristalsis of the esophagus with achalasia; however, in 2 of our patients who were studied peristalsis was normal, with the obstruction apparently due to a failure of the cardia to relax. Similar findings have been reported by others.

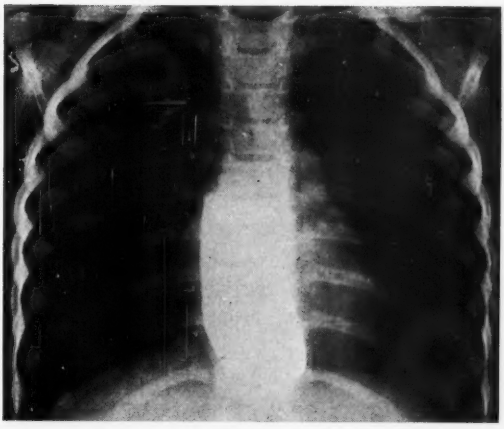


Fig. 10.—Case 4 before operation. Note the marked dilatation of the esophagus which ends in a blunt point at the cardia.

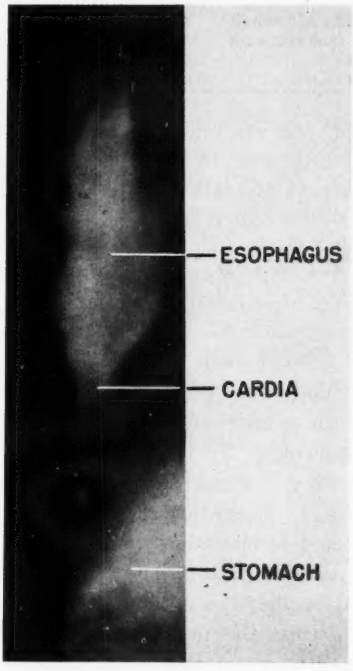


Fig. 11.—Same patient as in Fig. 10 2 years after surgery. The barium passed easily through the cardia to the stomach.

TABLE I. SUMMARY OF SIX CHILDREN WITH ACHALASIA

NO.	PATIENT	SEX	RACE	AGE (YR.)	CHIEF COMPLAINT	WEIGHT LOSS	FLUOROSCOPY FINDINGS	TREATMENT		RESULTS
								NUMBER OF DILATATIONS	SURGERY	
1	R. P.	F	W	1	Regurgitation	Yes	Slight dilatation	1	—	Improved
2	J. P.	F	W	2½	Regurgitation	Yes	Spasm of cardia, marked dilatation	6	Cardioplasty	Cure
3	C. B.	F	W	3	Regurgitation and vomiting	Yes	Slight spasm of cardia, moderate dilatation	3	—	Cure
4	M. McG.	F	W	4½	Dysphagia	Yes	Slight spasm of cardia	2	—	Cure
5	P. McF.	M	W	11	Dysphagia and vomiting	Yes	Spasm of cardia, moderate dilatation	2	—	Cure
6	J. B.	M	W	12	Chronic cough and vomiting	Yes	Spasm of cardia, marked dilatation	4	Cardioplasty	Cure

There is some similarity in dysfunction between this aganglionic esophageal lesion and Hirschsprung's disease. In both conditions the aganglionic segments exhibit spasm and failure of relaxation. The proximal esophagus and colon gradually become dilated and hypertrophied. As the dilatation becomes more pronounced, the peristaltic waves fail to be occlusive and the efficiency of the contraction as a propulsive mechanism decreases. Furthermore, as the dilatation becomes massive and chronic infection becomes established in the esophageal wall, peristalsis may become diminished or absent. This hypothesis is supported by absent peristalsis which has been reported.

It seems reasonable that prolonged use of dilatation is unwise. We have discarded dilatations when a series of three has proved ineffective and have resorted to surgical treatment.

Experimental work has demonstrated that in dogs denervation of the lower end of the esophagus results in cardiospasm and dilatation of the esophagus. In our 2 patients subjected to operation there were no ganglion cells. These bits of evidence indicate that the basic lesion may be a defect in parasympathetic innervation in the distal esophagus. After esophageal dilatation most patients are free of symptoms, despite the fact that esophageal enlargement persists. The difference is that there is no significant delay as the swallowed bolus passes through the cardia. Four of our patients were in this category. The 2 patients who were resistant to dilatation were treated surgically and have been relieved of their symptoms. These have no evidence of esophagitis.

SUMMARY

Achalasia or cardiospasm is a rare condition in children and there is little concerning the condition in this age group available in the medical literature. We have reviewed our experience with 6 cases.

Strip biopsies were secured from the lower esophagus in 2 patients and these contained no ganglion cells. Similar strips of tissue were removed from the normal esophagus of patients at postmortem examination and these contained numerous ganglion cells. Peristaltic studies were made in the 2 patients operated upon and these demonstrated good propulsive function in the esophagus.

The similarity of this condition to that in adults is indicated. Pathologic studies in adults have been made by several investigators and a degenerative lesion of Auerbach's plexus of the lower esophagus has been demonstrated. It is postulated that in children the defect is the same except that the lesion is congenital in origin.

The problem of diagnosis in this obscure condition is discussed. The effectiveness of dilatation in 4 cases and of surgery on the lower esophagus in 2 cases is given on the basis of 2 to 8-year follow-up studies.

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(For Discussion, see page 95)

OBJECTIVE EVALUATION OF SURGERY FOR HIATUS HERNIA AND ESOPHAGITIS

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HIATUS HERNIA and esophagitis have been the subject of much clinical and experimental investigation, yet there have been few attempts to devise an accurate means of determining objectively the presence and severity of esophagitis in a given patient and there have been almost no attempts to assess objectively the effect of either medical or surgical treatment on the underlying pathophysiology. Quincke[1] is credited with the first description of this disease in 1879. Since then, Winkelstein,[2] Wangensteen,[3] Boyd,[4] Ellis,[5] and Allison,[6] and many others[7-10] have made significant contributions to our knowledge of the basic mechanism of this disease. Notwithstanding these contributions, there has been a need for an accurate means of detecting esophagitis—particularly in its early forms—as well as a means of measuring the effect of surgery on the incompetent gastroesophageal junction. It is well known that the symptoms of esophagitis, which consist of epigastric and substernal burning and pain with regurgitation, can be mimicked by heart disease, disorders of the biliary tract, ulcer diatheses, and psychogenic disturbances. In reviewing the data on 204 patients operated upon for hiatus hernia and esophagitis it became obvious that it was not possible to determine the extent of underlying disease present preoperatively, and the effect of surgery on the changes present. It was further obvious that some of the poor results in surgery can be ascribed to the fact that the clinician confused the symptoms of heart disease or other disorders with those of esophagitis.

The weight of evidence favors the concept that the symptoms and pathologic changes of esophagitis are due to reflux of acid gastric juice, which produces peptic digestion of the mucous membrane of the terminal esophagus. It has been shown that peptic activity occurs in a pH range of from 1 to 5.[11] Peptic activity is maximal at a pH of 2.3, falls to 20 per cent at a pH of 4, and is absent at a pH of 5. Significant peptic activity occurs in the pH range of 1 to 4. A measurment of the pH in the terminal esophagus, therefore, gives one a direct objective assessment of the amount of peptic activity occurring in

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this location. Unfortunately, the most careful x-ray examination may fail to show regurgitation into the terminal esophagus. Even in those patients in whom regurgitation is demonstrated by x-ray, peptic activity may be absent because of achlorhydria or hypochlorhydria. Esophagoscopy often fails to show significant changes in the lining of the terminal esophagus, even in the presence of severe symptoms.

MATERIALS AND METHOD

With the above factors in mind, we are conducting studies designed to give the clinician an objective means of determining the presence or absence of peptic activity in the terminal esophagus. To date, 134 experiments have been performed on 105 patients. The study has consisted of a direct measurement of the pressure changes and the pH in both the normal subject and patients with esophageal disorders. For measurement of pressure, an open-ended, water-filled polyethylene catheter, 0.047 inch internal diameter, is attached to a Statham pressure transducer (Model P23-G)* which in turn is connected to a Sanborn recording machine. The transducer is located as nearly as possible on a level with the esophagus which we have found, by fluoroscopy, measures approximately 11 cm. from the back, with the subject in the supine position. For measurement of pH, a Beckman electrode is used. This is attached to a Beckman pH meter† which is attached to a Sanborn machine‡ for continuous recording of pH. The pH circuit is completed by placing the reference electrode in the patient's mouth. The exploring electrode and pressure tube when taped together are no longer than a No. 14 Levin tube. The two devices attached together are passed transnasally into the stomach. Forty-five minutes prior to being tested, the subject is given 50 mg. of Histalog§ intramuscularly. This is done to eliminate variables, such as transient hypochlorhydria. In the early tests, the electrodes were positioned fluoroscopically, 2 inches below the diaphragm. With experience it was noted the electrode could be accurately positioned in the stomach by relating its location to the pressure inversion point at which the positive intragastric pressure changed to negative intraesophageal pressure. With the electrode in the stomach, the gastric pH is determined and if the subject is found to be hypochlorhydric with a pH above 4, 200 c.c. of 0.1 N hydrochloric acid are given to insure the presence of acid so that reflux, if present, can be detected. For recording simultaneous intragastric and intraesophageal pressures, an additional polyethylene tube is passed into the stomach. For these simultaneous pressure measurements it is necessary to use a multi-channeled recording machine. We have employed the Grass machine.‖ The type of tracing obtained by this method is shown in Fig. 1. It can be seen from this tracing that there is a sharp pressure inversion point at the level at which the intragastric positive pressure becomes negative intraesophageal pressure as the sensing device is

*Manufactured by the Statham Instrument Company.

†Zeromatic pH Meter, Model 96, manufactured by Beckman Instrument Co., Fullerton, Calif.

‡Sanborn Twin-Viso, Model 60-1300, manufactured by Sanborn Co., Cambridge, Mass.

§Betazole, Lilly (kindly supplied by Eli Lilly & Co., Indianapolis, Ind.).

‖Grass Model 5 Polygraph, manufactured by The Grass Instrument Co., Quincy, Mass.

pulled cephalad. In all 17 normal subjects studied, the pressure inversion point coincided sharply with the conversion of the pH from a level within the peptic range in the stomach to a level well outside the peptic range in the terminal esophagus. With the normal subject in the supine position, various respiratory and other maneuvers were performed. These included the Mueller (Fig. 2), the Valsalva maneuver, simulated hiccups, coughing, inspiratory and expiratory sniffs, as well as complete relaxation. In no normal subject, with the pH electrode placed 2 to 4 cm. above the pressure inversion point, were we

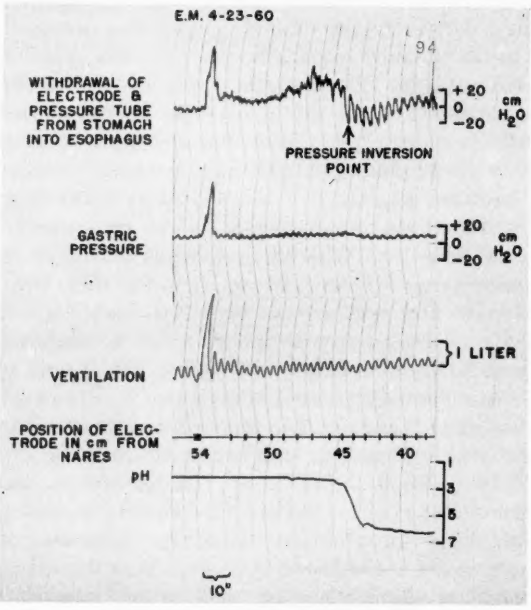


Fig. 1.—pH and pressure tracing on normal subject. Note the pressure inversion point (PIP) at which the positive gastric pressure changes to negative esophageal pressure. The PIP coincides with a change in the pH from a peptic level in the stomach to a level outside the peptic range in the esophagus.

able to produce reflux of acid gastric juice into the terminal esophagus, indicating that under the conditions of our experiments the gastroesophageal junction is a highly competent barrier against the reflux of acid gastric juice. This phenomenon can be repeated over and over again in the same subject by simply passing the sensing device up and down, across the pressure inversion point. These studies confirm the impression of Allison, and others, that the gastroesophageal mechanism is remarkably efficient in the normal individual in preventing reflux of gastric juice into the terminal esophagus. The pressure inversion point has been located in the average-sized individual at around 40 to 45 em. from the nares. The sensing device is calibrated so that, as it is pulled cephalad, the distance of the tip of the electrode from the pressure inversion point can be measured accurately. By this means, the height of regurgitation into the terminal esophagus can be precisely determined. This

appears at present to be a more accurate means of determining reflux than x-ray examination. In 78 patients in whom we were able to make an x-ray correlation with pH and pressure studies, 19 subjects were found in whom there was a failure of correlation between the presence of peptic activity in the lower esophagus and the x-ray findings. In 9 of these patients, after every maneuver had been attempted under fluoroscopy, the radiologist stated that there was no regurgitation and yet peptic activity was detected in the lower esophagus. This correlated, in each case, with the symptoms present. Ten of these patients had small hiatus hernias diagnosed by x-ray studies and the radiologist stated that the patient should show regurgitation, even though it

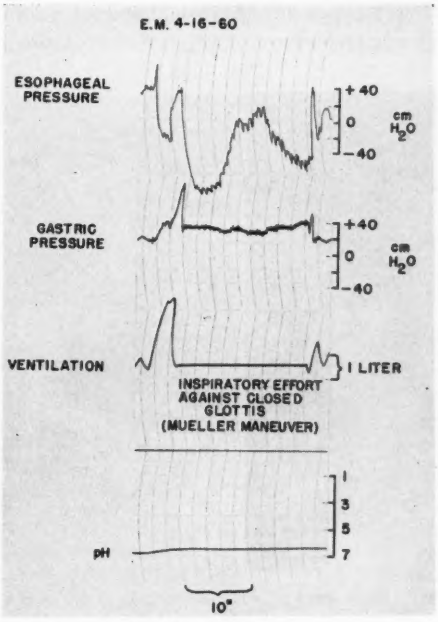


Fig. 2.—Mueller maneuver in normal subject. Note no change in the pH with this strong respiratory maneuver with the sensing device 4 cm. from pressure inversion point.

TABLE I. PRESSURE AND pH STUDIES (103 PATIENTS)

Normal	20
Patients with reflux	15
Patients without reflux	23
Preoperative patients	30
(with regurgitation 28)	
(without regurgitation 2)	
Postoperative patients	26
(with regurgitation 2)	
(without regurgitation 24)	
Recurrent hiatal hernia or ? of recurrence	6
Parahiatal hernia	2
Achalasia	4
Jejunal interposition	1
Short esophagus	4
Total	131

was not demonstrated at the x-ray examination. Our studies showed no evidence of peptic activity in the terminal esophagus in this group of patients. The studies done to date are shown in Table I. It is noted that a variety of esophageal disorders have been tested. These will form the subject of a later report.

REFLUX WITH NO DEMONSTRABLE HIATUS HERNIA

In contrast to the normal individual, patients with symptoms of esophagitis and those patients with demonstrable evidence of hiatus hernia almost invariably show the presence of peptic activity in the terminal esophagus. Fig. 3 illustrates a patient with symptoms of severe mid-sternal pain. This patient had been seeking medical advice because of the severity of this pain and had been

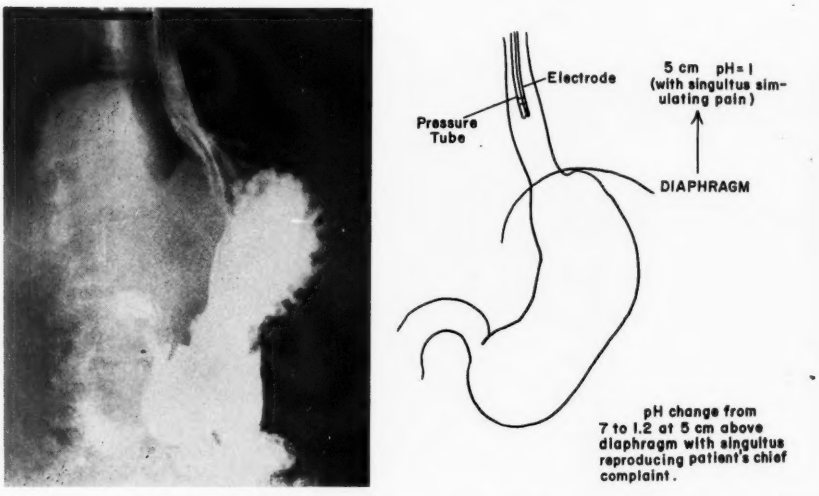

Electrode
5 cm pH= 1
(with singultus simulating pain)
Pressure Tube
DIAPHRAGM
pH change from 7 to 1.2 at 5 cm above diaphragm with singultus reproducing patient's chief complaint.

Fig. 3.—Subject with no hiatus hernia, with substernal pain. Pain reproduced following singultus.

told by several clinicians that he had heart disease, yet his electrocardiogram was not conclusive. His gastrointestinal x-ray studies showed only a widened hiatus with no hernia and no reflux. After a complete and thorough medical examination, the internist was at a loss to explain the patient's symptoms. With the patient in the supine position and with the sensing device 5 cm. above the diaphragm, several respiratory maneuvers were performed. After completing a series of simulated hiccups the pH in the terminal esophagus changed abruptly from a level around 5 to a pH of 1, well within the peptic range. Approximately 30 seconds later, the patient volunteered that he was having the type of pain in the mid-sternal region for which he had been seeking medical advice. This, then, graphically demonstrated the cause of the patient's pain and indicated that he was suffering from reflux into the terminal esophagus, even though no hernia was demonstrable. The patient has been placed on a

medical regimen. We have had the opportunity of studying 6 additional patients in whom no demonstrable hiatus hernia was present by x-ray examination but who had symptoms of esophagitis and who showed the presence of peptic activity in the terminal esophagus. These studies have proved to be a valuable adjunct in clarifying these difficult diagnostic problems.

THE SMALL SLIDING HIATUS HERNIA

In the group of patients with small hiatus hernias, one again encounters some of the most difficult diagnostic problems in this area of surgery. In our experience this has been the most common form of hiatus hernia. Of the 204 patients analyzed in our surgical material, 133 were classified as having small hiatus hernias. An example of this group of patients is shown in Fig. 4,

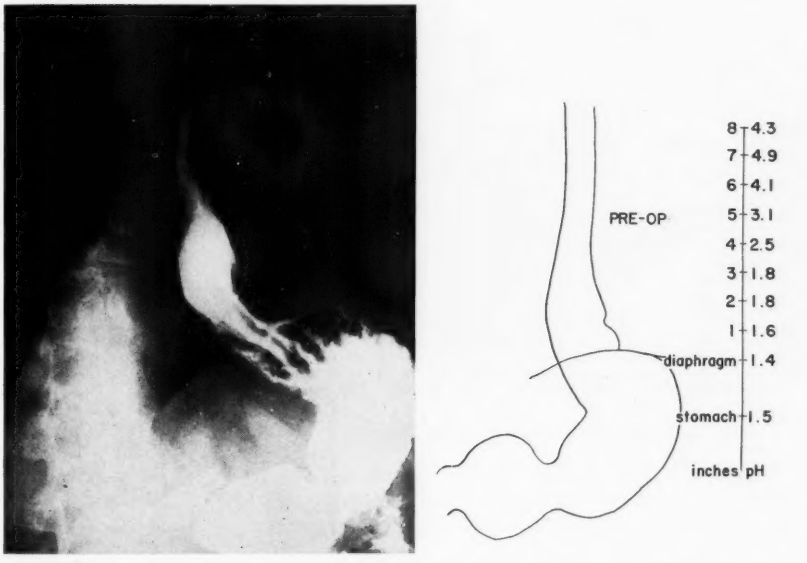


Fig. 4.—Preoperative study on patient with a small hiatus hernia whose symptoms suggested angina pectoris.

which illustrates a subject who had severe pain which could not be separated clinically from the pain of heart disease. The gastrointestinal series showed no regurgitation. A pH and pressure tracing was obtained, showing reflux almost to the upper third of the esophagus. Because of the severity of symptoms and the failure to respond to medical management, operation was performed. A postoperative study on the same patient is depicted in Fig. 5. It is noted that the pH changes sharply from a level well within the peptic range in the stomach to a level well outside the peptic range in the esophagus at the pressure inversion point. All respiratory maneuvers failed to produce regurgitation in this patient postoperatively. In effect, his tracing was indistinguishable from that obtained in the normal subject.

Of the 133 surgical patients with small hiatus hernias, pH studies were done preoperatively in 16, all of whom showed reflux. Postoperative studies were obtained on 14 of these. Thirteen showed complete correction of reflux, 1 patient showed transitory reflux after forceful inspiratory sniffing. This latter patient had severe symptoms preoperatively and is practically asymptomatic since operation. It should be noted that 10 patients with small hiatus hernias who were studied with the intraesophageal electrode, showed no reflux. These patients have not, as yet, been operated upon.

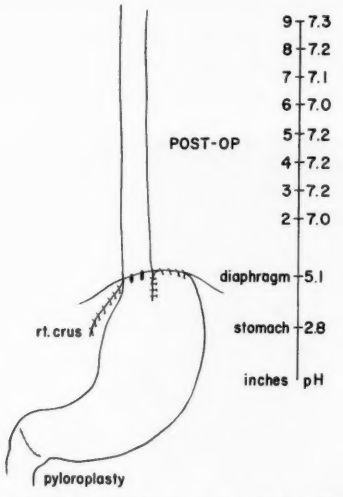


Fig. 5.—Postoperative study following repair of small hiatus hernia, showing correction of reflux above pressure inversion point. Patient was relieved of symptoms.

THE LARGE SLIDING HIATUS HERNIA

In the group of large hiatal hernias, we have arbitrarily included those which contained a fourth or more of the stomach in the thorax and which, at operation, readily admitted four fingers through the esophageal hiatus. In our material, there were 56 such patients. We have had an opportunity to study 9 patients with large hiatus hernias. Eight of these showed peptic activity with free regurgitation into the upper third of the esophagus. An example of this group is shown in Fig. 6. In this patient, peptic activity could be detected well above the gastric pouch dislocated into the chest. In these individuals care must be taken to be certain that the peptic activity is not simply confined to the gastric pouch dislocated above the diaphragm. By accurate measurement it can be clearly shown that the peptic activity continues well above the dislocated gastroesophageal junction in these patients. The presence of esophagitis was confirmed by esophagoscopy in 6 of these patients. However, it is surprising how minimal the observable changes in the terminal esophagus may be, in the presence of severe incapacitating symptoms of esophagitis. Thickening and reddening of the mucosa, with increased longitudinal folding,

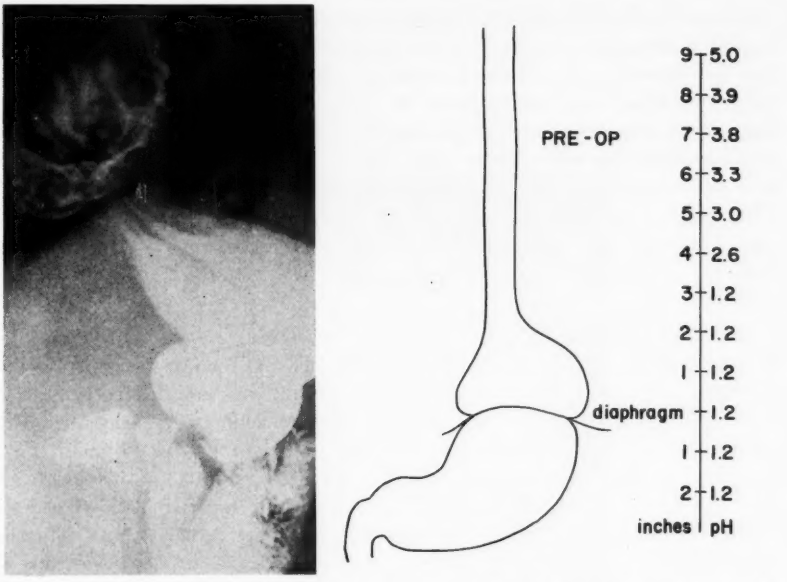
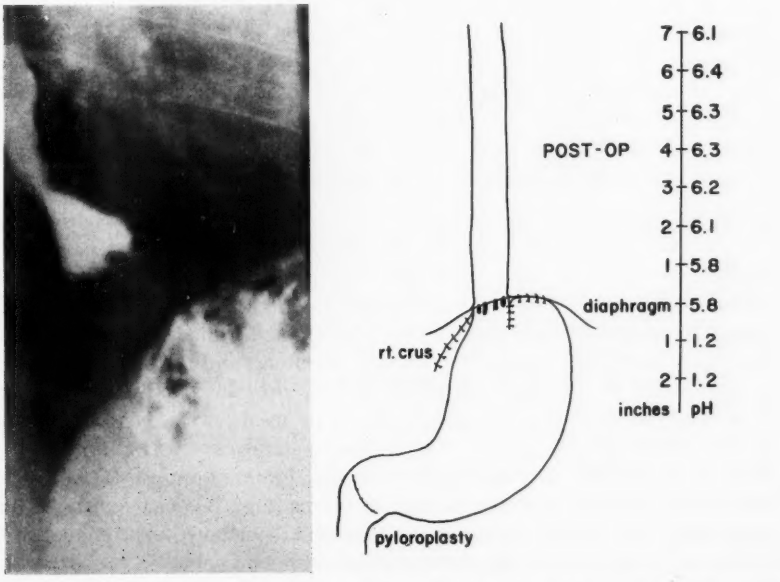


Fig. 6.—Preoperative study on patient with a large hiatus hernia, showing peptic activity to 8 inches above the pressure inversion point.



ig. 7.—Postoperative study following repair of a large hiatus hernia. pH and pressure findings were returned to normal and symptoms were relieved.

may be all that can be detected in the presence of long-standing severe symptoms. In the patient depicted in Fig. 6, medical management failed and operation was performed. At operation, the large hiatus hernia was repaired by the same method as used in patients with small hiatus hernias. A postoperative tracing on this patient (Fig. 7) showed an abrupt change in the pH at the pressure inversion point, from a pH well within the peptic range in the stomach to a pH outside the peptic range in the esophagus. She has been completely relieved of symptoms. The postoperative studies on 8 of the patients with large hiatal hernias show that reflux has been corrected in all of these and their tracings, at present, are indistinguishable from those obtained in the normal subject.

In 1 patient with a large hiatus hernia, which included up to one half of the stomach in the chest, pH studies demonstrated no peptic activity in the terminal esophagus. It was found that this patient maintained a gastric pH around 3.5 to 4, in spite of Histalog. Being hypochlorhydric, she obviously did not have a strong mechanism for the production of peptic esophagitis. Close questioning disclosed that she had no symptoms relative to this large hernia, thus indicating that the mere presence of a large hiatus hernia of the sliding variety is not necessarily an indication for surgical intervention. This hernia was repaired only because the patient had a carcinoma of the lower lobe of the left lung. It should be added at this point that demonstrable regurgitation alone, in the presence of hypochlorhydria, is not an indication for surgical intervention. Moreover, the presence of peptic activity in the terminal esophagus, as demonstrated by these studies, is also, in itself, not necessarily an indication for surgery. As with any other clinical test, the results must be correlated with clinical symptoms. Symptoms must be significant and refractory to medical management before surgery is recommended for hiatus hernia and esophagitis.

PARAESOPHAGEAL HERNIAS

True paraesophageal hernias have been uncommon in our experience and of the 204 patients operated upon only 7 were classified as having paraesophageal hernias of significant size. We have had an opportunity to study two paraesophageal hernias. One of these is depicted in Fig. 8. This huge hernia contained the entire stomach and the first portion of the duodenum inverted into the thorax. This patient had severe pressure symptoms and had bled repeatedly over a long period of time and was found to have profound anemia. Her symptoms were those of crushing substernal pain after meals, with dyspnea, palpitation, and, at times, protracted vomiting. Preoperatively she was studied and found to have no evidence of peptic activity in the terminal esophagus. At operation, the entire stomach was found in the thorax and it was confirmed that the esophagus was well maintained in its normal position by strong attachments posteriorly, consistent with a well-defined phreno-esophageal membrane. Careful dissection showed the gastroesophageal junction to be well below the diaphragm. The hernia was repaired and the patient has been entirely relieved of her symptoms. In contrast to the sliding type of hiatus hernia, in which

esophagitis is the prime cause of symptoms, in the paraesophageal hernia, incarceration of the viscus in the thorax is the most important cause of difficulty. In both patients studied by pH and pressure determinations, a competent gastroesophageal sphincter was found. In 1 additional patient with a huge paraesophageal hernia, incarceration and strangulation of the stomach in the

Fig. 8.—Parahiatal hernia with entire stomach and first portion of duodenum inverted into chest. No reflux could be demonstrated, but severe symptoms due to incarceration were present.

thorax produced severe vomiting which led to profound water depletion and electrolyte imbalance which was nearly fatal. At operation, the stomach was reduced from the hernia and was found to be viable. The hernia was repaired and the patient recovered and has been free of symptoms for 5 years.

SURGICAL TECHNIQUE

The type of operation that has been utilized in both the small and large hiatus hernia is depicted in Fig. 7 and consists of reduction of the stomach below the diaphragm, closure of the leaves of the right crux of the diaphragm, reconstruction of the gastroesophageal angle, and approximation of the fundus of the stomach to the undersurface of the diaphragm. This type of repair has been utilized in most patients within the last 4 years. Prior to that time, a number of patients were treated by simple closure of the hiatus. An analysis

of these patients shows that in 125 operations reconstructing sutures were utilized in addition to closure of the hiatus. In this group there have been only 2 verified recurrent hernias in the past 6 years. In the 68 patients in whom no reconstructing sutures were utilized, there have been 7 verified recurrent hernias, all of whom are symptomatic. We have studied 2 patients in whom only simple closure of the hiatus was performed and free regurgitation into the esophagus was demonstrated. One of these has been repaired twice, with simple closure

Fig. 9.—Severe stenosing esophagitis with long segment of narrowing.

of the hiatus and no additional reconstructing sutures. Following the second repair he continues to demonstrate free regurgitation into the upper third of the esophagus and has severe symptoms. The 2 patients with recurrent hernias in whom reconstructing sutures were employed, in addition to closure of the hiatus, have not had symptoms sufficient to warrant re-operation and we have not had an opportunity to study these patients by pH and pressure determinations. One must infer, from both the clinical and experimental evidence, that simple closure of the hiatus is an inadequate operation for hiatus hernia and esophagitis. It is noteworthy that in all of the patients with sliding hernias and paraesophageal hernias it was possible to retract the stomach below the diaphragm, even in the presence of severe long-standing esophagitis. This is contrasted with the situation in patients with short esophagus.

HIATUS HERNIA AND STENOSING ESOPHAGITIS

This group of patients present the most difficult problems in management. Severe stenosing esophagitis with shortened esophagus has been an unusual occurrence in our experience and we have encountered this in only 4 patients. All 4 of these patients were completely obstructed and all 4 had bled significantly and had profound anemia. Two types of stenosing ulceration occurred in these patients. One, illustrated in Fig. 9, shows a long segment of extreme narrowing. This patient was completely obstructed preoperatively, in spite of repeated attempts at dilatation. The other type of severe esophagitis is

Fig. 10.—Severe stenosing esophagitis with discrete marginal ulceration.

shown in Fig. 10, which illustrates the typical discrete marginal ulceration. This discrete ulceration occurred at the junction of the gastric and esophageal mucous membrane. All 4 of these patients were found to have a hiatus hernia. Preoperative pH studies in all 4 patients invariably showed a high gastric acidity with a pH in the range of 1 to 2. Prior to the onset of obstruction, reflux well into the upper third of the esophagus could be demonstrated. When obstruction occurred, reflux, of course, ceased beyond the point of obstruction. All 4 of these patients had been subjected to dilatation repeatedly and were refractory to medical management. Operation became mandatory because of obstruction, severe depletion, as well as repeated hemorrhage. At operation it was obviously impossible to retract the stomach below the diaphragm, even after extensive freeing of the esophagus. In these patients, the thickened,

inflamed esophagus was found to be adherent to the pericardium, the pleura, and surrounding structures, including the arch of the aorta. Even after dividing these dense adhesions, little additional mobility could be obtained. In all 4 patients, we have utilized the procedure described by Ellis,[12] consisting of resection of the stenosed segment along with resection of the antrum (Fig. 11). The vagotomized and antrectomized stomach pouch is then anastomosed to the cut into the esophagus. This is followed by gastrojejunostomy. To this procedure we have added a jejunojejunostomy in order to increase the food

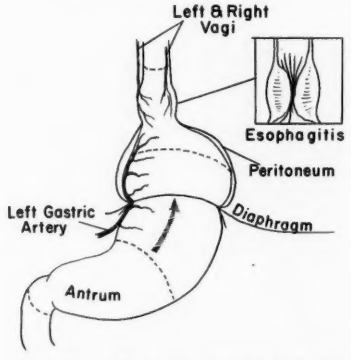


Fig. 11.—Severe stenosing esophagitis with hiatus hernia and short esophagus. Dotted lines show lines of resection of stenosed segment and antrum.

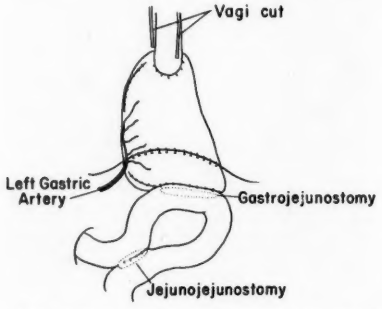


Fig. 12.—Completed modified Ellis operation for severe esophagitis with short esophagus.

reservoir and to further prevent alkaline reflux into the gastric pouch and thence into the esophagus. All 4 patients survived the operation nicely. One patient died 1 month after operation, of pneumonia. She was 74 years of age and was operated upon as a last resort because of complete obstruction and bleeding. She was severely depleted and in borderline uremia at the time of operation. In all 4 patients, including the patient who subsequently died, postoperative studies have shown a pH in the gastric pouch between 6 and 7, with the exception of 1 patient in whom a small central area was located in the pouch in which the pH was around 2.5. By esophagoscopy and by pH studies, there was no peptic activity in the terminal esophagus in any of

the patients. In the patient who shows a gastric pH in the peptic range, the absence of esophagitis is undoubtedly due to the small volume of gastric juice which is neutralized by saliva from above and alkaline jejunal content from below. None of these patients have symptoms of regurgitation or esophagitis postoperatively. The first of these operated upon has been followed for 1½ years. The completed operative procedure is shown in Fig. 12.

DISCUSSION

The need for a method of detection and objective assessment of the severity of esophagitis is obvious. A measurement of the pH in the terminal esophagus gives the surgeon an accurate estimate of the presence and severity of peptic activity in this area. These studies have been of value as a diagnostic tool in those patients in whom x-ray has failed to demonstrate either a hiatus hernia or regurgitation, but who are suffering with severe symptoms. We feel that this is particularly important because it is in this group of patients with no demonstrable hernia or with a small hernia in whom many diagnostic errors are made. It is in this group that many of the poor surgical results obtained in the past have occurred, since the symptoms in these patients may be confused with a number of other disorders. These studies have also been of value in assessing the effect of surgical treatment upon the underlying pathophysiology. In those patients who had an adequate operation and who were studied postoperatively, complete correction of the tendency to reflux could be demonstrated. In those patients with only simple closure of the hiatus and no additional sutures to reconstruct the gastroesophageal angle, or to hold the stomach in its position below the diaphragm, continued regurgitation was demonstrated, either clinically or experimentally, in 7 patients, indicating that simple closure of the hiatus is an inadequate procedure for hiatus hernia and esophagitis.

Moreover, studies which are now in progress to determine the relationship between gastric and esophageal pressure and the presence of a pressure gradient across the gastroesophageal junction should do much to elucidate the normal and abnormal mechanisms of the esophagogastric junction, an area about which there is much to learn.

SUMMARY

1. The need for a simple method of determining the presence or absence of esophagitis in a given patient is obvious.

2. Studies (135) consisting of simultaneous measurement of pH and pressure in the area of the esophagogastric junction have been performed on 104 subjects in our laboratory.

3. These tests have become an important diagnostic tool, especially in the patient who has marked symptoms but in whom no hiatus hernia or reflux is demonstrable by x-ray.

4. These studies have been valuable in assessing the effect of surgery on the underlying mechanisms of hiatus hernia and esophagitis.

5. Both clinical and experimental evaluation of operations for hiatus hernia show that simple closure of the hiatus is an inadequate operation for patients with esophagitis.

6. Contiuation of these studies, and particularly differential gastroesophageal pressure studies, should do much to elucidate further the mechanisms of the esophagogastric junction.

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(For Discussion, see page 95)

INSTRUMENTAL PERFORATION OF THE ESOPHAGUS

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EVEN in the hands of expert endoscopists, an occasional instrumental perforation of the esophagus occurs. This complication has been associated with an exceedingly high mortality and morbidity.

The importance of early operation in the pre-antibiotic era was emphasized by Neuhof and Jemerin.[4] In a series of 40 patients, 16 of 28 who were operated upon survived, whereas only 2 of the 12 patients who were not operated upon survived. All operations were drainage procedures and closure of the perforation was not attempted in any patient. In a later paper, Jemerin[1] noted an increase in the number of cases of esophageal perforations and ascribed the rise to the more frequent use of endoscopic examinations. He attempted to evaluate the influence of sulfadiazine and penicillin on the results of treatment. He concluded that the marked improvement in results in his later cases was due to more frequent early operation and that antibiotics should be used only as an adjunct to prompt operation.

Samson[7] in 1951 and Weisel[9] in 1952 advocated immediate surgical intervention and primary repair of the perforation rather than surgical drainage alone. Weisel described excellent results in 7 patients so treated. Overstreet and Ochsner[5] in 1955 reported 13 cases of perforation of the esophagus and emphasized the importance of immediate surgical repair. They made no differentiation between the treatment of cervical and intrathoracic perforations of the esophagus.

In a series of 24 cases reported by Rietz and Werner,[6] in 1958, were 7 patients with cervical perforations; 4 of these died, all owing to infection. None of the 7 patients was treated by primary closure because of the long delay before the patients were seen by the surgeon and the serious condition of the patients on admission to the hospital. In addition to the usual drainage procedures, all these patients received antibiotics. These authors concluded that a recent perforation of the esophagus should be regarded as a surgical emergency and treated as any other perforation of the alimentary tract. Because an abscess did not form when antibiotics were used in 1 patient who had sustained

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a perforation of the cervical esophagus, Jones[2] suggested that chemotherapy allows a delay in undertaking the "formidable procedure" of mediastinotomy. Korkis[3] also recommended antibiotic therapy as the initial treatment for perforation of the cervical esophagus. He advocated operation only when such therapy failed, as evidenced by suppuration or abcess formation.

In discussing the treatment of instrumental perforations of the esophagus, Terracol and Sweet[8] state: "The advent of antibiotic medication to combat infection has greatly altered the seriousness of the condition and the method of treatment. In fact, many cases of esophageal perforation which formerly

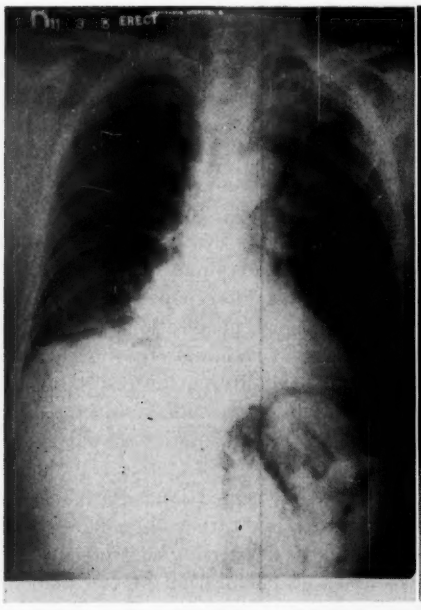

Fig. 1.

Fig. 2.

Fig. 1.—Case 1. Roentgenogram made immediately after gastroscopy showing mediastinal emphysema and air beneath the diaphragm.

Fig. 2.—Case 1. Lipiodol study of esophagus, 17 days after thoracotomy, which shows false passage in upper esophagus.

would have been attended by a fulminating infection and a fatal outcome, nowadays can be expected to follow a benign course ending in healing without surgical intervention. In many instances, however, both antibiotics and surgical treatment must be employed. As in all other situations antibiotics cannot supplant the drainage of abscesses and the removal of offending foreign bodies."

In the light of these conflicting opinions it seemed desirable to review our experience with the treatment of instrumental perforation of the esophagus. From November 1952 to August 1958, members of the surgical staff of the Jefferson Medical College Hospital were asked to treat 18 patients who had sustained an instrumental perforation of the esophagus. Summaries of the histories of these patients follow.

CASE REPORTS

CASE 1.—C. M., a 48-year-old man, entered the hospital Oct. 20, 1952, because of gastrointestinal symptoms and anemia. A gastroscopic examination was performed on Nov. 3, 1952. Immediately following the examination he developed subcutaneous emphysema over the anterior chest wall and up into his right cheek. A diagnosis of esophageal perforation was made and he was transferred to the surgical ward. On arrival he had marked subcutaneous emphysema of his chest, neck, and face. A chest roentgenogram (Fig. 1) revealed extensive subcutaneous emphysema and a large amount of air beneath the left diaphragm. No definite mediastinal emphysema was noted. He was given antibiotics, a whole blood transfusion, and intravenous fluids. Three and one-half hours after the perforation, the patient was operated upon through a left posterolateral thoracotomy. A large rent in the posterior wall of the esophagus at the cardioesophageal junction was found.

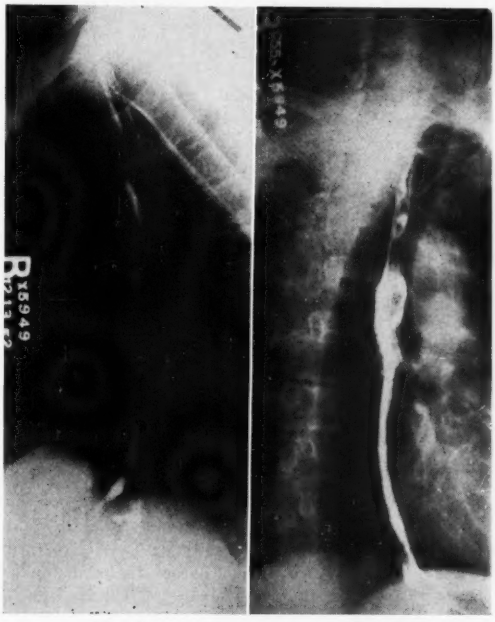

Fig. 3. Fig. 4.

Fig. 3.—Case 1. Lipiodol study of esophagus made 39 days after thoracotomy. There is narrowing and shortening of the blind pouch.

Fig. 4.—Case 1. X-ray study of esophagus made 2½ years after thoracotomy. Normal study.

The nasogastric tube passed preoperatively could be seen in the rent. An attempt to pass this tube into the stomach was unsuccessful because it was in a plane between the mucosa and muscular wall of the esophagus. The muscular layer was divided up to the arch of the aorta, but it was not possible to see the hole in the mucosa. The Levin tube was withdrawn and reinserted through the lumen of the esophagus into the stomach. The rent in the muscular coat of the lower esophagus and stomach was repaired and the mediastinal, pleural, and periotoneal spaces drained. The early postoperative course was complicated by retained pulmonary secretions requiring bronchoscopy and by auricular fibrillation which was controlled with quinidine and digitalis. The esophagus was visualized with Lipiodol on Nov. 20, 1952. The roentgenogram revealed a long false passage beginning at the upper end of the esophagus and ending blindly at the level of the mid-thoracic esophagus (Fig. 2). On the twentieth postoperative day, feedings were started through a nasogastric tube which was

left in place for 19 days. X-ray studies on December 12 revealed marked shortening and narrowing of the blind pouch (Fig. 3). Oral feedings were begun on that date and he was eating without difficulty when discharged on Dec. 26, 1952. No diverticulum could be seen in the esophagus in roentgenograms made 2½ years later (Fig. 4).

Comment: Failure to find and close the laceration of the mucosa and closure of the muscular coat of the esophagus created a blind pouch, which necessitated the prolonged use of a nasogastric tube for feeding. Prompt operation and drainage of the mediastinum certainly obviated other serious complications.

CASE 2.—P. H., a 57-year-old man, entered the hospital on Feb. 14, 1953. Five hours before admission the lower end of his esophagus had been dilated by bougienage in the Out-Patient Department for benign cicatricial stenosis. One hour after the dilatation he experienced severe, generalized, abdominal pain associated with frequent belching. On admission his temperature was 97.3° F., pulse 88, and blood pressure 130/80 mm. Hg. He appeared acutely ill and he held himself rigid with the hips flexed. There was marked tenderness, rebound tenderness, and muscle guarding in the upper abdomen. Peristalsis was absent. X-ray studies of the chest and abdomen revealed no abnormalities other than an enlarged heart. It was believed that he had a perforation of the gastrointestinal tract just below the diaphragm. Parenteral fluids and massive doses of antibiotics were begun. Within 2 hours of admission his temperature rose to 100° F., the pulse rate to 104, and the blood pressure to 180/100 mm. Hg. Five hours after admission a laparotomy was performed through an upper midline abdominal incision. The peritoneal cavity was normal except for the presence of multiple stones in the gall bladder. The incision was then extended into the chest by splitting the sternum and dividing the diaphragm. This revealed a mediastinitis with food and gastric contents in the posterior mediastinum. The esophageal hiatus was three times normal size, and there was a constricting band of circular muscle fibers about 3 cm. above the cardioesophageal junction. A perforation could not be found although the esophagus was explored to the level of the aortic arch and a finger was placed within the esophagus by way of a gastrotomy. A Heller-type myotomy was performed and the esophageal hiatal hernia repaired. A nasogastric tube was passed into the stomach. Two intercostal drainage tubes were placed in the right pleural cavity, leaving the mediastinal pleura on the right widely open.

The next day, Feb. 16, 1953, he developed auricular fibrillation which was controlled by increasing the digitalis he had been receiving for many years. On the fourth day the upper chest drainage tube was removed. On the fifth day the temperature rose to 103° F. On the tenth day, serosanguineous fluids drained from the abdominal wound which had partially dehisced. On the thirteenth day open drainage of a large right empyema was done. Despite this drainage, the patient died 3 days later.

Comment: Because of a mistaken diagnosis of peritonitis, an exploratory laparotomy was performed. When no peritonitis was found, extension of the wound into the thorax by means of a median sternotomy and diaphragmatic incision gave an inadequate exposure of the lower esophagus and the perforation was not found.

The cause of death was sepsis from an overwhelming empyema which was recognized too late.

CASE 3.—L. B., a 57-year-old man, was admitted to Jefferson Hospital on Feb. 15, 1953, because of inability to swallow solid foods. The following morning esophagoscopy was undertaken. Because of a language barrier, the patient was quite uncooperative. The esophagoscopist reported that during the attempt to pass the scope, it was introduced beneath the mucosa of the esophagus just below the cricopharyngeus muscle and tunneled in this layer for about 5 cm. He did not think that the muscular layer of the esophagus was perforated. The procedure was abandoned before he was able to visualize the lesion.

Immediately following esophagoscopy, the patient was quite comfortable. A roentgenogram of the neck revealed a "small amount of free air in the retropharyngeal space dissecting laterally toward the left." X-ray studies of the chest showed full expansion of the lungs and no demonstrable air in the mediastinum (Fig. 5). The patient was started on parenteral fluids and antibiotics in large doses and allowed nothing by mouth. That evening and the following day the patient complained of slight soreness along the left side of the neck. Two days after the esophagoscopy, x-ray examination with Lipiodol revealed an annular constriction at the junction of the middle and lower thirds of the esophagus extending for about 5 cm. There was no evidence of a fistula and no Lipiodol was seen in the soft tissues of the neck.

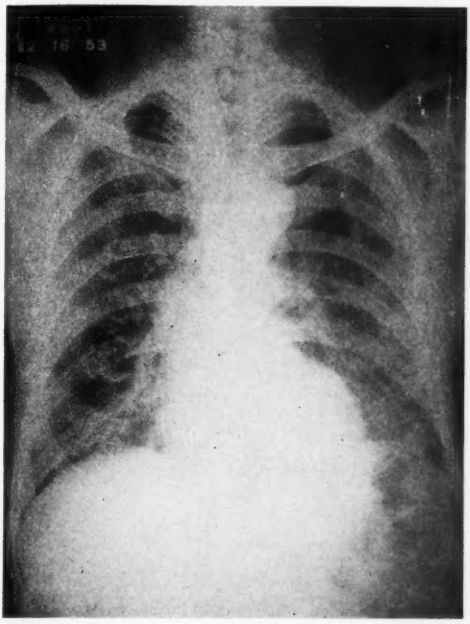

Fig. 5.—Case 3. Chest roentgenogram made after esophagoscopy. The film was normal except for a small amount of air in the neck.

On Feb. 20, 1953, 4 days after perforation, the patient was operated upon. Despite the fact that mediastinal emphysema was not seen on the x-ray film, there was considerable air throughout the mediastinum. A carcinoma of the middle third of the esophagus was resected and an esophagogastrostomy performed. On the day after operation, a tracheostomy was done because of profuse tracheobronchial secretions. Oral feedings were begun on the fourth postoperative day. He was discharged 14 days after operation, at which time he was able to eat a soft diet.

Comment: Whereas x-ray studies revealed only a slight amount of air in the neck and none in the mediastinum, there proved to be a considerable amount of air throughout the mediastinum at operation. Thus the absence of demonstrable air in the mediastinum on a chest roentgenogram does not rule out mediastinal emphysema.

CASE 4.—H. R., a 45-year-old woman, was found to have a low gastric acidity during examination at a cancer detection center. For that reason she was gastroscoped as an out-

patient on Feb. 26, 1953. The report is as follows: "At 9:00 A.M., the gastroscope was passed with ease until the cardia was reached and here an impassable obstruction was encountered which was considered to be due to spasm. Gentle pressure and insufflation were attempted. The patient became very restless, but no evidence of shock or unusual complaints of pain followed. It was noted that swelling about the right eye was pronounced." The gastroscopy was terminated and when the drapes were removed from the patient, it was evident that she had pronounced subcutaneous emphysema from her hips to her eyes. She was transferred directly from the out-patient department to the surgical ward.

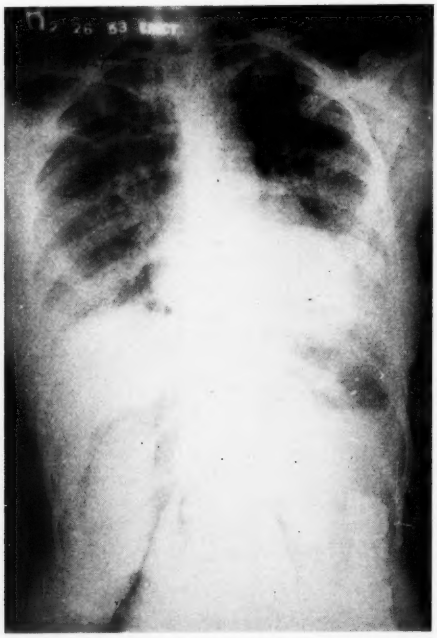

Fig. 6.—Case 4. X-ray study made immediately after gastroscopy showing extensive air throughout the retropleural and retroperitoneal tissues.

On admission to the ward she was apprehensive but did not complain of pain. An x-ray study of the chest and abdomen (Fig. 6) demonstrated marked subcutaneous emphysema and extensive insufflation of air throughout the retroperitoneal tissue. The initial decision was to treat this patient nonoperatively. However, during the next few hours her condition grew rapidly worse. Reluctance of the patient caused further delay, but she was finally operated upon 10 hours after perforation. One hundred and fifty milliliters of turbid fluid were found in the left pleural cavity. The mediastinum was opened widely allowing air and fluid to escape. Three centimeters above the diaphragm a longitudinal rent, 2.5 cm. long, was found in the esophagus. The perforation was closed with interrupted catgut sutures. The mediastinum and pleural space were drained with a fenestrated tube attached to an underwater seal. The patient was started with oral feedings on the fifth day. An intact esophagus was demonstrated by a barium swallow on the twelfth postoperative day and the patient was discharged 3 days later.

Comment: The deterioration of this patient's condition with nonoperative treatment was quite dramatic. Fortunately, this occurred so rapidly that the initial decision was promptly reversed and the patient was operated upon 10

hours after the perforation. The patient's smooth subsequent course is an example of the type of convalescence possible when early operation and closure of the perforation is carried out.

CASE 5.—H. R., a 51-year-old man, had a diagnostic bronchoesophagoscopy performed on May 7, 1953. Bronchoscopic findings were all within normal limits. Esophagoscopy disclosed a narrowing of the lower esophagus with much spasm, but nothing was observed to suggest neoplasm. One hour later he complained of severe pain in the left flank and became quite dyspneic. His temperature was 101° F. and pulse was 100. The trachea was deviated to the right. Hyperresonance was present, breath sounds were inaudible over the left hemithorax and the apex beat of the heart was shifted to the right. Chest x-ray studies (Fig. 7) disclosed a left pneumothorax and a small amount of fluid at the base of the left lung. A spontaneous pneumothorax rather than a perforated esophagus was thought to be the problem.

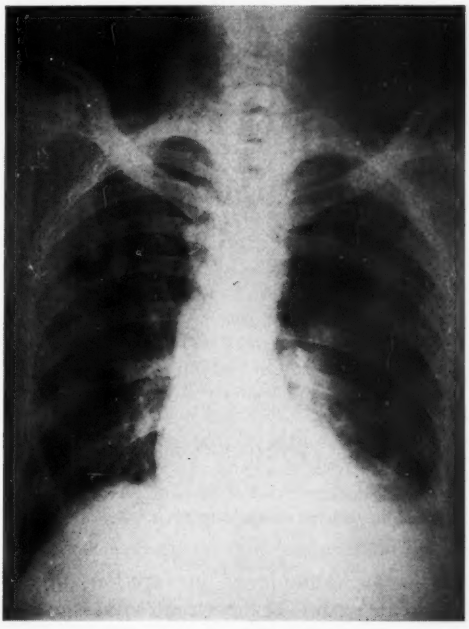

Fig. 7.—Case 5. Chest roentgenogram made immediately after esophagoscopy which shows left pneumothorax.

A thoracentesis in the left second interspace anteriorly produced adequate decompression and marked relief of the patient's symptoms. Parenteral fluids and antibiotics were given and a nasogastric tube passed for feeding purposes. The following day, May 8, 1953, a repeat x-ray study of the chest revealed a smaller pneumothorax on the left but an increase in the fluid at the left base to the eighth rib in the posterior axillary line. The patient continued to run a septic course with temperatures ranging between 101° and 103° F. On May 14, 1953, 7 days after the bronchoesophagoscopy, a Lipiodol swallow revealed a perforation of the lower end of the esophagus, 5 cm. above the diaphragm. A closed drainage of the left pleural cavity was performed and copious amounts of thick, foul pus were obtained. The following day, open drainage of a large abscess cavity was performed. The patient continued to do poorly. Four days later a tracheostomy was required. The patient became disoriented, developed signs of pericarditis, and died 19 days after perforation and 12 days after the initial chest drainage.

Comment: Instruments which perforate the esophagus may also perforate the mediastinal pleura with resultant hydropneumothorax. The delay of 1 week in establishing the correct diagnosis could have been avoided by a simple x-ray examination with Lipiodol. The delay in instituting proper treatment was obviously responsible for this patient's death.

CASE 6.—J. R., a 3-year-old girl, drank a solution that was presumed to contain lye. She sustained burns of her face, lips, and the inside of her mouth, as well as of the esophagus. She was admitted to another hospital, treated symptomatically, and discharged 6 days later, eating a soft diet.

One month later the child was readmitted to the same hospital because she was unable to eat. She was regurgitating saliva and had lost considerable weight. Two days later, on Nov. 11, 1953, she was transferred to Jefferson Hospital. At esophagoscopy the following day, a narrowing of the lumen of the esophagus was encountered several centimeters below the cricopharyngeus muscle. It was possible to pass a small bougie to the level of the diaphragm, but the endoscopist was not certain whether the bougie entered the stomach. About 6 hours following the procedure, the child had a rectal temperature of 102° F. and a pulse of 130. Examination revealed crepitus in the left supraclavicular area. Breath sounds were audible bilaterally with dullness to percussion at the base of the left lung posteriorly. The abdomen was diffusely tender with muscle guarding in the epigastrium. X-ray studies of the chest and abdomen demonstrated a perforation located on the left side at the level of the ninth dorsal vertebra. The child was started on parenteral fluids and large doses of penicillin and streptomycin. On Nov. 13, 1953, approximately 28 hours after perforation, a right posterior mediastinotomy was performed through the eighth and ninth ribs extrapleurally. Large amounts of air and fluid were evacuated from the mediastinum and two drains were inserted. On Nov. 14, 1953, a tracheotomy was performed for retained secretions. On Nov. 18, 1953, a feeding gastrostomy was performed. The patient improved slowly and on Dec. 3, 1953, a repeat examination of the esophagus by x-ray failed to demonstrate a fistula. On Dec. 7, 1953, she was allowed sips of water by mouth, and a string was passed through the mouth and out the gastrostomy tube. On the following day, retrograde dilations of the esophagus were begun. The last dilation was in August, 1954, and since that time she has been able to eat any type of food.

Three years later at the age of 6, this child was again seen at Jefferson Hospital after she had swallowed a coin which lodged in her esophagus. This was removed through an esophagoscope on May 16, 1956, and a roentgenogram of the esophagus made at that time showed it to be normal throughout.

Comment: This is an example of perforation during dilation of the esophagus. The 28-hour delay between perforation and operation increased the morbidity by allowing mediastinitis to become well established and precluding primary closure.

CASE 7.—E. O., a 48-year-old woman, was esophagoscoped on July 23, 1954, at another institution for removal of a meat fragment in the lower esophagus. The following week she had an intermittent fever and complained of pain up and down her back. Following treatment with antibiotics, the fever and pain subsided and she was discharged on Aug. 3, 1954, able to eat soft food, but she still complained of a sore throat. Nine days later she was readmitted with chills, fever, and severe pain on swallowing. A barium swallow revealed a high perforation of the esophagus with barium in the mediastinum. At this point she was transferred to Jefferson Hospital.

On admission, on Aug. 14, 1954, the temperature was 103° F., pulse 144, respirations 34, and blood pressure 190/110 mm. Hg. She coughed continually bringing up barium and large amounts of mucus. She appeared dehydrated and her neck was swollen bilaterally. X-ray

studies (Fig. 8) revealed a superior mediastinal abscess with an air pocket at the level of the lower two cervical vertebrae. Barium (Fig. 9) was seen in the mediastinum, trachea, and bronchi. After rapid rehydration, a right cervical mediastinotomy was performed 4½ hours after admission. No attempt was made to locate or close the perforation. A small nasogastric tube was inserted for feeding purposes. Within 24 hours the pulse and temperature had returned to normal. The patient was maintained on tube feedings for 25 days, at which time both x-ray examination and esophagoscopy showed that the fistulous tract had closed. She was able to eat without difficulty and was discharged from the hospital on Sept. 15, 1954, 32 days after operation and 54 days after the original esophagoscopy.

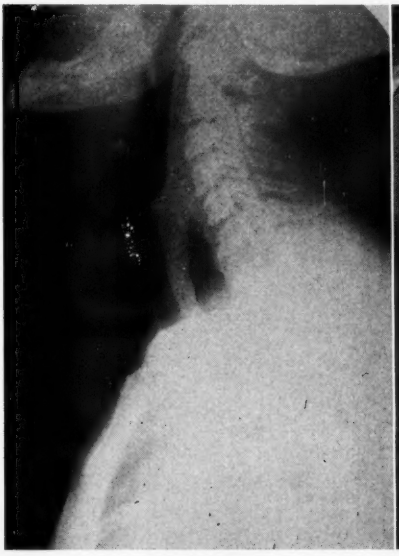

Fig. 8.

Fig. 9.

Fig. 8.—Case 7. Roentgenogram of neck made 22 days after esophagoscopy which demonstrates air pocket.

Fig. 9.—Case 7. X-ray examination of chest made 22 days after esophagoscopy. There is barium in the mediastinum on the tracheobronchial tree.

Comment: This patient is one of the fortunate few who was able to survive prolonged mediastinitis. This is probably due to the fact that the abscess remained localized in the neck. Had immediate operation been performed, a prolonged morbidity might have been averted. No effort was made to close the perforation because of the condition of the local tissues at the time of operation.

CASE 8.—A. P., a 69-year-old man, was admitted to Jefferson Hospital on Jan. 12, 1955, because of dysphagia after swallowing an orange seed. He was esophagoscoped on the day of admission and the scope was passed with difficulty. No foreign body was encountered, but there was a narrowing of the esophagus from the cricopharyngeus muscle to below the aortic arch. Immediately following the procedure, the patient complained of pain in the back of the chest and subcutaneous emphysema was noted in the neck. Penicillin and streptomycin were started and a feeding catheter was placed in the esophagus. Surgical consultation was not requested and immediate x-ray studies were not made. On the second hospital day

he fell and injured his left hip. A roentgenogram of the hip revealed a fracture of the left femoral neck. A chest roentgenogram (Fig. 10) taken at this time demonstrated subcutaneous emphysema in the soft tissues of the neck and hydropneumothorax with partial collapse of the right lung and a fluid level at the tenth rib posteriorly. The patient was slightly disoriented, but afebrile. Closed drainage of the right pleural cavity was established through a tube in the seventh interspace at the mid-axillary line and 800 ml. cloudy yellow fluid was evacuated along with considerable air. The patient continued to do poorly, with large amounts of fluid draining from his chest. He developed a markedly irregular heart beat which was treated with quinidine, and he became progressively more disoriented. On the suggestion of the medical consultant, the patient was transferred to the surgical ward on Jan. 18, 1955, 6 days after the perforation. On arrival the patient was acutely ill with a temperature of 101° F. and a pulse of 110. There were no breath sounds over the right

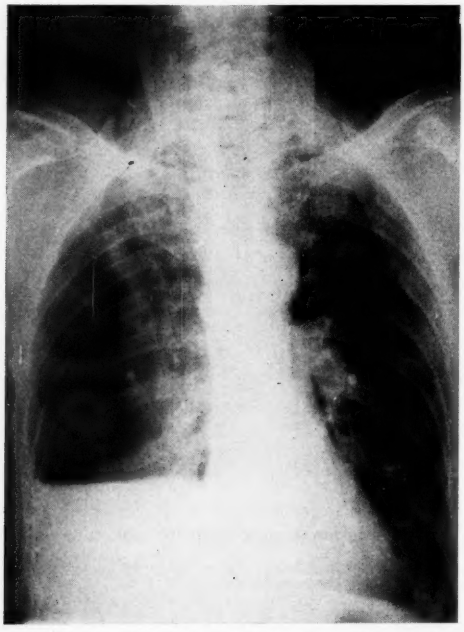

Fig. 10.—Case 8. Roentgenogram of chest made 2 days after esophagoscopy showing left hydropneumothorax.

lung and the percussion note was flat. The following day a tracheostomy was performed to facilitate removal of tracheal secretions. Following this, respiratory and cardiac symptoms improved. On Jan. 27, 1955, under continuous spinal anesthesia, the left hip was reduced and fixed under direct vision. The patient seemed to improve slightly; however, the empyema continued to drain pus as well as gastric contents. On Jan. 31, 1955, under local anesthesia, a Stamm gastrostomy was performed for feeding purposes. The feedings from this spilled out through the chest drainage. Although drainage of the empyema seemed adequate, he continued a downhill course and died on Feb. 2, 1955. Significant findings at autopsy were three lacerations of the esophageal mucosa in the right posterior wall at the level of the aortic arch, with dissection for 4 cm. distally and 3 cm. proximally, and extensive tearing of the serosa. There was focal mediastinitis and right empyema, as well as three acute gastric and two acute duodenal ulcers.

Comment: Failure to diagnose promptly and failure to promptly institute adequate treatment undoubtedly were largely responsible for this patient's death.

CASE 9.—G. G., a 28-year-old man, was admitted to Jefferson Hospital for the third time on Feb. 8, 1955. He had had an esophagogastrostomy for achalasia in 1948 and a subtotal gastrectomy for bleeding gastric ulcer in 1950. Four hours prior to the present admission, the patient had had an esophageal dilatation by bougienage in the out-patient department. Shortly before admission he was suddenly seized with severe constant pain in his left upper abdominal quadrant. This was accompanied by pain in the left lower chest that was exaggerated with respirations and radiated to both shoulder tops.

On admission the temperature was 101°F., respirations 36, pulse 96, and blood pressure 110/80 mm. Hg. He had respiratory distress and there was a suggestion of crepitus in the left supraclavicular area. The left lung field was dull to percussion, with distant breath sounds and moist râles at the base. Tenderness was present in the left upper abdominal quadrant, with muscle guarding and absent peristalsis. X-ray studies of the chest and abdomen revealed marked elevation of the left hemidiaphragm with a gas collection beneath it. There was no evidence of mediastinal emphysema and no abnormal clouding of the lung fields. Approximately 11 hours after perforation, a left exploratory thoracotomy and exploration of the peritoneal cavity through the diaphragm were performed. No perforation of the gastric pouch or esophagus could be found. In order to pass a Levin tube through the strictured esophagogastric junction into the gastric pouch, cardioplasty was performed. The diaphragm was closed and the mediastinum drained transpleurally by an intercostal tube attached to an underwater seal. On the fourth postoperative day, the nasogastric tube was removed and oral feeding was begun. He was discharged from the hospital on Feb. 21, 1955. An x-ray study on the day of discharge revealed that the material passed readily through the esophagus and entered the stomach without delay. One year later, May 3, 1956, the patient was readmitted to Jefferson for removal of an impacted bolus of meat from his lower esophagus. Esophagoscopy revealed a marked degree of esophagitis and superficial ulceration. Barium swallow showed a stricture of the esophagogastric junction. The patient has been followed subsequently with dilations at intervals of 3 months.

Comment: Despite early operation and a careful search for the perforation, it was never found. However, he responded to drainage of the area and feeding through a nasogastric tube. He unquestionably had had a perforation near the esophagogastric junction.

CASE 10.—J. Y., a 42-year-old man, was esophagoscoped at Jefferson Hospital on April 25, 1955, for study of an anemia of undetermined etiology. The esophagus appeared normal. However, during the procedure, bleeding was noted from high up on the posterior wall of the esophagus and a piece of tissue was found adherent to the esophagoscope. Following the procedure the patient experienced severe anterior chest pain and pain over the upper back. A high perforation was suspected. Parenteral fluids and antibiotics were started and a nasogastric tube inserted. Immediate x-ray examination of the chest revealed no evidence of mediastinal emphysema or mediastinitis. The consulting surgeon suspected perforation but he elected to observe the patient. The next morning the patient's temperature reached 101° F. with a pulse of 150. Lipiodol study at that time outlined a normal cervical and thoracic esophagus with no evidence of a fistulous tract. Medical, surgical, and ear, nose and throat consultants agreed that further nonsurgical treatment should be tried since the patient seemed somewhat better clinically than during the previous night. However, the septic course continued, and 9 days after esophagoscopy, x-ray examination revealed widening of the superior mediastinum. A posterior mediastinotomy was performed with drainage of 100 ml. of thick, foul pus. Subsequent x-ray studies showed a gradual narrowing of the superior mediastinum. The patient was discharged 21 days after operation, symptom free.

Comment: The failure to demonstrate a perforation of the esophagus by x-ray examination with Lipiodol resulted in a delay of 9 days in instituting the proper treatment.

CASE 11.—H. H., a 62-year-old man, was esophagoscoped at Jefferson Hospital on May 26, 1956, because of a 6 months' history of progressive difficulty in swallowing, associated with a weight loss of 20 pounds. A firm stenosis was found 37 cm. from the upper alveolar margin. There were fixation and ulceration of the entire circumference of the esophagus at that level. The esophagoscope could not be passed through the area of stenosis. A biopsy of the ulcerated area revealed squamous cell carcinoma, intermediate grade. Approximately 2 hours after the procedure the patient complained of chest pain, shortness of breath, and was found to be cyanotic. A diagnosis of traumatic perforation of the esophagus was made. Nine hours after the esophagoscopy the patient was operated on through a left thoracotomy. A left empyema and a necrotizing mediastinitis were found. The esophagus was perforated midway between the aortic arch and the diaphragm. There was a carcinoma in the esophagus extending from the lower border of the aortic arch to a point 5 cm. above the diaphragm. An esophagectomy and esophagogastrostomy above the aortic arch were performed. The mediastinum and left pleural space were drained with a fenestrated intercostal drainage tube placed posteriorly. Two days postoperatively, the patient developed auricular fibrillation and was digitalized. He made a good recovery and was discharged on June 8, 1956, 13 days postoperatively.

Comment: Early operation, with resection of the esophagus and esophagogastrostomy, even in the presence of an early empyema, achieved a good result in this case.

CASE 12.—P. G., a 20-month-old white baby girl, swallowed, at 7:15 A.M. on May 28, 1956, a hair clip which lodged in her esophagus. She was admitted to another hospital where an attempt at endoscopic removal was made at 11:30 A.M. Extraction was not possible because the point of the object had penetrated the left lateral wall of the esophagus and could not be freed. The patient was then transferred to Jefferson Hospital.

Upon arrival, the child was restless with a temperature of 105° F. and a pulse of 160. There was subcutaneous emphysema over the anterior chest. X-ray examination of the chest (Fig. 11) revealed a foreign body in the lower esophagus, with the point outside of the esophagus, and a small left pneumothorax. The patient was given parenteral antibiotics and intravenous fluids. At 10:00 P.M., 10½ hours after perforation, a posterolateral thoracotomy was made and a perforation of the lower esophagus identified on the left lateral wall. The foreign body was removed and the perforation closed with interrupted sutures of 5-0 chromic catgut. The mediastinum was left open for drainage and a tube was placed in the pleural space and attached to an underwater seal. After an uneventful recovery, the child was discharged on June 10, 1956.

Comment: An attempt to remove a foreign body by esophagoscopy resulted in perforation of the esophagus. Prompt operation and definitive treatment gave a good result.

CASE 13.—S. S., a 66-year-old woman, had an esophageal dilation as an out-patient on the morning of May 14, 1957, for a long-standing benign stricture. On the way home she experienced diffuse, crampy, abdominal pain, and sharp epigastric pain. She stopped to eat, thinking she possibly had hunger pains. On arrival home, her colicky abdominal pain continued and localized in the epigastrium and left upper quadrant. She also vomited 2 to 3 teaspoonfuls of dark, red blood. She had no dyspnea or anterior chest pain. She was advised to return to the hospital which she did at 10:15 P.M. On admission, the temperature was 102° F., pulse 100, respirations 22, and the blood pressure 140/90 mm. Hg. The lung

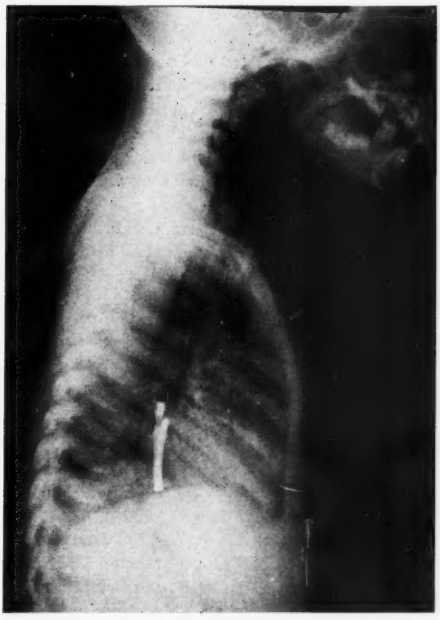

Fig. 11.—Case 12. Roentgenogram made after esophagoscopy which shows foreign body in esophagus.

Fig. 12.—Case 13. X-ray study made 10 days after dilation which demonstrates barium in the peritoneal cavity.

fields were clear. There was slight tenderness in the epigastrium and left upper quadrant but no rebound tenderness or rigidity. Peristalsis was normal. X-ray studies of the chest and abdomen were normal. Despite these negative findings, the surgical consultant thought that the patient had a perforation of the esophagus, and advised an x-ray examination of the esophagus using a contrast medium. This was not done. The following morning the patient was dehydrated, with a temperature of 100° F. and still complained of epigastric pain. X-ray examination of the esophagus with Lipiodol was again suggested but again was not performed. On May 16, 1957, the patient seemed somewhat better. The temperature had dropped to 99° F. and the pulse rate to 64. There was good peristalsis and she had a normal bowel movement. Repeat x-ray films of the chest and abdomen revealed no change from the day of admission. Still no x-ray study of the esophagus using a contrast medium was performed. The patient continued to improve except for an intermittent fever. Feeding through a nasogastric tube was started on May 17, 1957. On May 21, 1957, 7 days after admission, the tube was removed and oral feeding begun. Three days later, an x-ray study of the esophagus and stomach using a barium meal (Fig. 12) revealed a perforation of the gastrointestinal tract in the region of the esophagogastric junction. A large collection of barium was seen outside the stomach, apparently in the lesser omental sac. The patient was then transferred to the surgical service. The following day, on May 25, 1957, 11 days after esophagoscopy, the patient was operated upon through an upper midline abdominal incision. The perforation at the esophagogastric junction was located and closed. Necrotic tissue, barium, and old blood clots were removed from the lesser omental sac. Drains were placed in the lesser sac and supra- and subhepatic spaces. At this point in the procedure, the patient suddenly went into shock and the blood pressure could not be measured. It was necessary to use vasopressor drugs to maintain a blood pressure. The next morning, the patient was alert, although l-norepinephrine was still necessary to maintain blood pressure. Retained bronchial secretions, cardiac arrhythmia, and a pulmonary infarction then developed. On June 3, 1957, a tracheostomy was required because of continued difficulty with secretions. The following day, June 4, 1957, 21 days after admission and 10 days after operation, the patient died suddenly during aspiration via the tracheostomy.

Comment: This highlights a serious problem in the handling of these patients. In many instances, the surgeon has no difficulty in deciding the proper treatment but has difficulty in convincing the attending physician of the urgency of operation.

CASE 14.—L. D., a 46-year-old Negro woman, entered the hospital on Feb. 6, 1958, because of weakness, weight loss, and epigastric pain. She was known to have diabetes mellitus and cirrhosis of the liver for 3 years. The diabetes was out of control because she had not been taking her prescribed insulin. On Feb. 19, 1958, an upper gastrointestinal x-ray study revealed a shallow ulcer crater on the posterior wall of the distal body of the stomach. Six days later a gastroscopic examination showed "a shallow benign appearing ulcer on the lesser curvature at the incisura angularus." Institution of a medical ulcer regimen promoted healing of the ulcer crater as demonstrated on March 14 by a repeat gastrointestinal x-ray study. On March 15, 1958, at 9:00 A.M. a repeat gastroscopic examination was performed in order to ascertain the amount of healing that had taken place. The gastroscope passed without difficulty to a point several centimeters below the pharynx. An obstruction was encountered at that level and the examination terminated.

One half hour later she complained of belching and left chest pain with radiation to the neck. The temperature was 100° F. She was allowed nothing by mouth and intravenous fluids were started. Although the diagnosis of esophageal perforation and early mediastinitis was entertained by the intern, the physician in charge did not seek surgical consultation until the patient's condition became worse. Ten hours after gastroscopy, crepitus was noted in the neck, and the pulse rate and temperature had risen to 120 and 102.4° F., respectively. Approximately 11 hours after gastroscopy, a chest x-ray examination

of the esophagus with contrast medium and surgical consultation were requested. X-ray studies (Fig. 13) revealed a large amount of air in the soft tissues of the neck and a Lipiodol study (Fig. 14) showed a cervical esophageal perforation with extravasation of contrast medium to the left. She was given large doses of parenteral antibiotics. At 1:00 A.M. on March 16, 1958, 16 hours after gastroscopy, she was taken to the operating room and a left cervical mediastinotomy made. The superior mediastinum was filled with purulent material. A posterior perforation of the esophagus at the level of the sixth cervical vertebra was found and closed with interrupted chromic catgut sutures. Drains

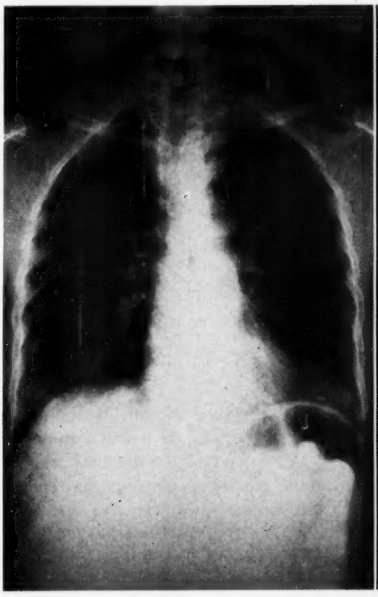

Fig. 13.

Fig. 14.

Fig. 13.—Case 14. Roentgenogram of chest made 11 hours after gastroscopy which demonstrates air in the neck.

Fig. 14.—Case 14. Lipiodol study which demonstrates perforation of the cervical esophagus.

were placed in the superior mediastinum, and a nasogastric tube inserted. The temperature and pulse returned to normal on the fourth postoperative day. However, because of difficulty in controlling her diabetes, supplemental intravenous fluids were continued for 7 days at which time a liquid diet was tolerated. She refused surgical treatment for her recurring gastric ulcer and was discharged on April 16, 1958, 1 month after operation.

On Oct. 21, 1959, the patient was readmitted with diabetic acidosis. The hemoglobin was 4.1 Gm. and hematocrit 15 per cent. Her diabetes was brought under control, she was transfused and, on Oct. 12, 1959, a subtotal gastrectomy for benign ulcer was done. She was discharged 16 days later, having made a good recovery from the operation.

Comment: Although there was an 11-hour delay in operation, closure of the perforation in the cervical esophagus gave a good result. This is an example of the physician in immediate charge of the patient failing to recognize the dire consequences following esophageal perforation until the patient's condition began to deteriorate.

CASE 15.—B. H., a 53-year-old man, swallowed a piece of meat which appeared to stick beneath the lower sternum. The next morning, on May 19, 1958, an unsuccessful attempt at endoscopic removal of the foreign body was made. Shortly thereafter a perforation was recognized. He was given morphine and transferred immediately to Jefferson Hospital. On arrival at 2:00 P.M. he was critically ill, in shock, markedly cyanotic with slow, shallow respirations, and a temperature of 102° F. Breath sounds were absent over the right hemithorax. The diagnosis of esophageal perforation with a right pneumothorax was made. In the receiving ward, a catheter was placed through the right second interspace anteriorly and the pneumothorax decompressed. A stab wound was made in the left side of the neck and the superior mediastinum decompressed. After this, respirations improved and the cyanosis regressed. He was then sent directly to the operating room where right thoracotomy, esophagotomy with removal of the foreign body, and extensive mediastinotomy with pleural drainage were performed. No perforation was found in the thoracic esophagus. Following closure of the thoracotomy wound, the patient was turned to the supine position and the incision in the left side of the neck which was made in the receiving ward was extended. The superior mediastinum was widely entered and fluid evacuated. A medium-sized rubber covered gauze drain was placed in the superior mediastinum and the wound closed loosely. He was much improved at the completion of the operation. The patient made a good recovery and was discharged on June 7, 1958.

Comment: This patient's condition rapidly deteriorated following perforation of the esophagus. He appeared moribund on admission to the hospital and the resuscitative measures in the accident ward probably were lifesaving. The interval between perforation and operation was not unduly long, which helps to explain his good recovery.

CASE 16.—B. J. B., a 29-year-old man, was admitted to Jefferson Hospital on Oct. 10, 1958, because of weakness and tarry stools for the previous 48 hours. He also complained of epigastric burning relieved by alkali. An upper gastrointestinal x-ray study revealed marked delay in passage of opaque medium past the esophagogastric junction with dilatation of the esophagus above this level. No evidence of ulcerative disease of the esophagus, stomach, or duodenum was noted. A small feeding tube was passed and continuous drip feeding begun. On Oct. 13, 1958, an attempt at esophagoscopy under local anesthesia was unsuccessful. On Oct. 16, 1958, esophagoscopy under Pentothal anesthesia was also unsuccessful because of a "small pharyngeal space." On Oct. 27, 1958, over a previously swallowed string, an esophagoscope was passed into the region of the cricopharynegeus muscle. At this point, a perforation of the esophagus was suspected and the examination terminated. Several hours later the patient began to complain of pain on swallowing and crepitus was noted in the left supraclavicular region. Immediate chest x-ray examination revealed air in the superior mediastinum with deviation of the trachea to the right. He was started on large doses of parenteral antibiotics, intravenous fluids, and given nothing by mouth. Twenty-four hours later his temperature had risen to 102° F. and the pulse to 140. The patient was then operated upon under general endotracheal anesthesia and the superior mediastinum drained through a cervical incision. No attempt was made to close the perforation in the esophagus. A persistent salivary fistula prolonged his hospital course. He was discharged 19 days after operation, able to eat soft foods.

Comment: Nonoperative treatment of this perforation of the cervical esophagus was persisted in for 26 hours. This precluded primary closure of the perforation, even though it was easily identified, and prolonged his convalescence.

CASE 17.—M. P., a 74-year-old woman, entered the hospital at 2:00 A.M. on May 23, 1959, because of syncope which followed gastrointestinal bleeding. She had been under medical care for a hiatal hernia, labile hypertension, and peripheral vascular disease.

She was given blood transfusions and started on a Sippy diet with alkalies. On May 24, 1959, an upper gastrointestinal x-ray study revealed a paraesophageal hiatal hernia and an active gastric ulcer. On June 3, 1959, at 11:00 A.M. she was gastroscoped in an effort to visualize the gastric ulcer and hiatal hernia. She had marked thoracolumbar scoliosis and the examination was unsuccessful, the scope reaching a point just below the larynx before being withdrawn. Trauma to the esophagus was not suspected. Three hours later she complained of epigastric pain that was aggravated by breathing. She was very pale and dyspneic. There was bilateral swelling of the neck with palpable crepitus. A diagnosis of an esophageal perforation with mediastinal and subcutaneous emphysema was made and surgical consultation requested. A chest x-ray study (Fig. 15) revealed mediastinal emphysema extending into the soft tissues of the neck and atelectasis of the right lung

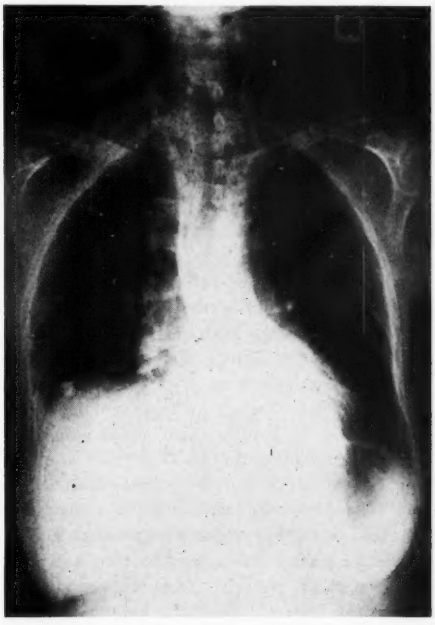

Fig. 15.—Case 17. Roentgenogram of chest which demonstrates marked mediastinal emphysema.

at the base posteriorly. Examination of the esophagus with contrast material revealed a perforation on the right just below the cricoid cartilage. At 4:00 P.M. 5 hours after perforation, the patient was operated upon through a right cervical mediastinotomy. The esophageal perforation was identified and repaired and the superior mediastinum drained. The patient was then placed on her left side and a right posterior mediastinotomy was made at the level of the mid-thorax. Quantities of air were evacuated from the posterior mediastinum and a rubber-covered gauze drain introduced into the posterior mediastinum. The patient tolerated the procedure well. She was discharged from the hospital on the twenty-eighth day, able to eat a full diet.

Comment: The abnormal curvature of the spine contributed to the perforation by the gastroscope. Although the perforation occurred in the cervical area, the surgeon liberated considerable air and fluid when he wisely drained the mediastinum.

CASE 18.—B. M., a 70-year-old woman, was admitted to another hospital on Aug. 22, 1959, because a piece of meat had become impacted in her esophagus the day before. After a barium swallow, esophagoscopy was performed in an unsuccessful attempt to remove the impacted meat. Four hours following esophagoscopy the patient complained of severe chest pain and became dyspneic and orthopneic. Chest x-ray studies revealed a left pneumothorax, and thoracentesis was performed for decompression. The next morning an attempt was made to pass a nasogastric tube into the stomach but a chest roentgenogram showed that the tube had entered the left pleural space. Consequently it was removed. She was then permitted hourly sips of water in an attempt to wash the retained barium from the esophagus. On Aug. 24, 1959, a tube was passed into the esophagus and the barium evacuated. Because her condition was deteriorating she was transferred to Jefferson Hospital where she arrived at 8:00 P.M., Aug. 24, 1959.

The patient was acutely ill, markedly dehydrated, and moderately cyanotic. The temperature was 100° F., pulse 120, and blood pressure 130/90 mm. Hg. The respiratory rate was 32 per minute, with obvious prolongation of expiration. There was subcutaneous emphysema in the neck and over the thorax and the trachea was deviated to the right. There was slight hyperresonance at the base of the left lung and a definite decrease in breath sounds over the left hemithorax. The heart revealed a sinus tachycardia of 120 with frequent premature contractions. Chest x-ray examination revealed considerable air in the left pleural space with a moderate amount of fluid at both lung bases. She was given intravenous fluids, parenteral antibiotics, intravenous aminophylline and Solucortef, and was placed in an oxygen tent. After 12 hours of preparation, she was taken to the operating room. When she was placed on the operating table, auricular fibrillation developed. Operation was delayed for 2 hours while the fibrillation was treated with parenteral Gedilanid and quinidine which reduced the heart rate to 130 per minute. A left thoracotomy revealed several hundred milliliters of thin, cloudy material. The posterior mediastinum contained about 150 ml. of pus which lay anterior to the esophagus. The perforation in the esophagus could not be located. Through an esophagotomy a mild narrowing was noted at the diaphragmatic level of the esophagus which would admit only the tip of the index finger. There was esophagitis throughout the distal esophagus. Several fragments of putrid meat were removed from the esophagus and the esophagogastric junction was dilated with a finger. A nasogastric tube was introduced into the stomach, and the empyema and mediastinal abscess were drained. The day after operation she showed marked improvement. However, on the second postoperative day the temperature and pulse began to rise, and increasing tracheal secretions required frequent nasotracheal aspirations. Breath sounds were depressed over the base of the right lung and markedly depressed over the middle and lower lung fields on the left. Death occurred 43 hours after operation.

Autopsy revealed lacerations of the posterior wall of the upper one third of the esophagus, fibropurulent posterior mediastinitis, abundant mucoid secretions within the bronchi with marked atelectasis of the right lower and right middle lobes, left fibrinous pleuritis with acute congestion and edema of the left lung, bilateral pleural effusions, and fibrinous pericarditis.

Comment: The delay of 2 days in instituting proper treatment was certainly the major factor in the death of this patient.

RESULTS

The ages of the patients ranged from 20 months to 75 years. There were 10 men and 8 women. The cervical esophagus was perforated in 7 patients and the intrathoracic portion of the esophagus in 11 patients. The perforation

was produced by an esophagoscope in 10 patients, 5 of these during diagnostic esophagoscopy and 5 during the removal of foreign bodies. The gastroscope and bougie were each responsible for four perforations.

All patients in this series were operated upon. The interval between perforation and operation ranged from 3½ hours to 22 days. The surgical procedures ranged from simple drainage to esophagectomy. Thirteen of the 18 patients survived, a mortality of 28 per cent.

Operation was undertaken within 24 hours of perforation in 10 patients. Only one of these patients died (Case 2). Eight patients were operated upon more than 24 hours following perforation. Only 4 of these survived. Three of these 4 had sustained a perforation of the cervical esophagus (Cases 7, 10, 16). The fourth patient was operated upon 28 hours after perforation of the intrathoracic esophagus (Case 6).

The perforation was closed in 6 of the 10 patients who were operated upon within 24 hours and all 6 survived. Closure was attempted in only 1 of the 8 patients in whom operation was delayed more than 24 hours. This patient was operated upon 11 days after the perforation. She died several days later and it is doubtful that the closure held (Case 13). Four patients died of the 11 in whom only a drainage procedure was performed. A major factor in three of these deaths (Cases 2, 5, 8) was an overwhelming empyema, and in the fourth case a mediastinitis (Case 18).

Of the 13 survivors, 6 had primary closure of the perforation or resection of the diseased portion of the esophagus. Four of these patients left the hospital 13 to 15 days after operation. Two had prolonged hospital stays, 1 because of a long false passage within the wall of the esophagus (Case 1) and the other because of severe diabetes mellitus (Case 14). Seven of the surviving patients were treated by drainage alone. One patient (Case 9) left the hospital 13 days after an exploratory thoracotomy and transpleural drainage of the mediastinum. The average postoperative hospital stay among the other 6 was 24 days.

In the surviving patients, the most frequent postoperative complications were retained bronchial secretions and auricular fibrillation. Tracheostomy was required in 2 patients. The disturbances of cardiac rhythm were treated with digitalis or quinidine.

The 8 patients who were operated upon after 24 hours had all been treated with antibiotics. Despite this, infection, empyema or peritonitis, played an important role in the four deaths. The four survivors all required drainage between 28 hours and 22 days after perforation. A posterior mediastinotomy was necessary in 1 patient and a cervical mediastinotomy in the other 3. This experience tends to confirm what Jemerin said 12 years ago " . . . chemotherapy should be used as an adjunct to prompt surgery."[1]

DISCUSSION

Instrumental perforation of the esophagus should not be difficult to diagnose if the possibility is borne in mind. In some cases, the endoscopist may

suspect the possibility of perforation during the procedure. This was true in 3 of the 4 cases of gastroscopic perforation. Since gastroscopy requires the insufflation of air, the subcutaneous emphysema accompanying a perforation may be very dramatic.

The early clinical picture varies, depending upon the site of perforation. A patient sustaining a cervical perforation may complain only of slight discomfort in the neck. Palpation may demonstrate some subcutaneous emphysema and a lateral x-ray view of the neck will show air in the tissue planes.

Occasionally patients with intrathoracic esophageal perforations show little reaction at first, but usually they develop systemic manifestations quite rapidly. The symptoms in our patients were mainly those of mediastinitis and empyema. Case 15, only 5 hours after perforation, appeared moribund on admission and his survival was attributed to immediate, judicious decompression of a pneumothorax in the emergency room. After an initial decision for nonoperative treatment, the rapid deterioration of Cases 4 and 10 resulted in an immediate reversal of thinking and early operation.

Sometimes perforation in the lower esophagus may suggest an intra-abdominal emergency. The mistaken impression that the perforation in Case 2 was below the diaphragm resulted in an abdominal incision; the subsequent extension of this incision gave an inadequate exposure and probably contributed to the fatal outcome. When necessary, the site of perforation can be outlined by the use of Lipiodol or Gastrografin. Barium should not be used because after its escape from the esophagus it acts as a foreign body in an infected area. Furthermore, in 2 of our patients (Cases 7 and 18), barium was aspirated into the tracheobronchial tree.

Some prefer to temporize with cervical perforations. We do not agree with this. While none of our patients who sustained cervical perforations died, the morbidity was higher in those who had delayed operation and drainage only. Furthermore, it is difficult preoperatively to ascertain the extent of the perforation. In Case 3 the x-ray film of the chest was not unusual, but at operation there was considerable air throughout the mediastinum. In other cases (15 and 17), while the esophageal perforation was in the neck, the infection extended into the thorax requiring a thoracic mediastinotomy.

It is not always easy to convince the physician in charge of the patient of the need for immediate operation. This is especially true when the esophageal perforation produces minimal signs and symptoms. When the diagnosis is made, all oral feedings should be stopped, antibiotics begun, nasogastric suction instituted, and immediate operation performed. Experience with the 18 patients reported here leads us to conclude that, whenever possible, immediate operation with closure of the perforation gives the best results.

SUMMARY

1. Eighteen patients who sustained an instrumental perforation of the esophagus are reported.

2. Five of the 18 died; a mortality rate of 28 per cent.

3. Mortality was affected by (a) site, (b) type of operation, and (c) time between perforation and operation. (a) The five deaths occurred among the 11 patients with intrathoracic perforations. (b) The perforations were closed primarily in 7 patients. One of these patients, with closure 11 days after perforation, died. There were four deaths among the 11 patients who had only drainage without closure. (c) One death occurred in the 10 patients operated upon within 24 hours after perforation. There were 4 deaths in the 8 patients operated upon after that time.

4. Prompt operation and closure of the perforation is recommended.

We are grateful to Dr. George J. Willauer for permission to include 2 of his private cases, and Dr. William T. Lemmon for allowing us to include 1 of his private cases in this series.

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DISCUSSION

(PAPERS BY SPENCER [PAGE 1], DIVELEY [PAGE 23], SWENSON [PAGE 49], HILL [PAGE 60], NEALON [PAGE 75], AND THEIR ASSOCIATES).

DR. RALPH D. ALLEY, Albany, N. Y.—First, I would like to stress the special pathologic features of traumatic aneurysm which distinguishes it from the more common aneurysms of arteriosclerotic and syphilitic origin. The patient with traumatic aneurysm is characteristically young, free of diffuse vascular disease, and the aneurysm arises from a transverse disruption of the intima-media of the aorta. There is no loss of substance. Hence, at least occasionally, it should be possible to restore aortic continuity without the use of a graft. The feasibility of this therapeutic concept in the surgical management of aneurysm of traumatic origin is illustrated by a case I wish to report.

(Slide) This familiar diagram shows the characteristic location of the aneurysm, the left atrial-femoral artery bypass now customarily employed, and restoration of aortic continuity following excision of the aneurysm by end-to-end anastomosis. The patient was a 21-year-old man who was brought to our attention 3 months after an automobile accident. His only symptom was a murmur of growing intensity which was audible to him and to his family.

(Slide) Here is the preoperative aortogram.

(Slide) This aortogram slide taken a half second later demonstrates the aneurysm more clearly. Note that there has been a complete circumferential disruption of the intima-media with a 6 cm. distraction of the two ends.

(Slide) This aortogram taken a month postoperatively shows no trace of the anastomotic site.

The concept of restoring aortic continuity by direct suture is not new. Here I must point out the only flaw in Dr. Spencer's excellent presentation. One of the cases listed in his series in the category of tangential excisions was a middle-aged woman whose traumatic aneurysm was excised by Dr. Bahnson in 1952 employing endoaneurysmorrhaphy, effecting a closure of the neck of the sac by a transverse suture line. This patient is alive and free of recurrence. On the other hand, in the reported cases of tangential excision and placement of the suture line in the long axis of the aorta the patients either died of recurrence and rupture or required a secondary procedure. The rationale of aortic reconstruction in the same axis as the pathogenetic intima-medial tear is clear. The feasibility of accomplishing this, even in the presence of a complete disruption of these layers, is demonstrated by our case.

We urge, therefore, that the present custom of treating aneurysms of traumatic origin by excision and grafting be modified by exploration in each case of the possibility of excision and restoration of aortic continuity by direct suture.

DR. WILLIAM W. HEROY, Huntington, N. Y.—I rise to describe an 18-year-old patient who was admitted to the emergency room and whom I saw subsequently, with the following picture of acute rupture.

This particular individual showed over a period of the next half hour increasing numbness of the lower extremities, with increasing pain in the legs, followed by numbness. About an hour after he arrived in the accident room, without any drop in blood pressure, without any blood in his left chest, there was a line of demarcation at about the level of his umbilicus with cold, pale skin from there on down, and no motor power in his legs.

We took him to the operating room, induced anesthesia, and rapidly opened up the left chest through a posterolateral approach. About the time we opened the chest the heart went into standstill, and on attempting massage, it was very obvious that as I squeezed the heart, it would not fill in anywhere near the normal rate. It was necessary to wait for the heart to fill which it did slowly before I could give it the next squeeze.

This precluded our being able to accomplish what we were trying to do, and he died.

At autopsy, the spinal cord was not injured and it was interesting to find that the hematoma, instead of being limited to the mediastinum, had descended down the extraperitoneal space, extending down below the inguinal ligaments, and he had, in effect, cross-clamped his aorta at about the level of the diaphragm.

I think this is rare, but I think that hematomas may present a real problem. Even if you are able to handle the problem in the upper portion of the aorta, often you may have to decompress the hematoma below, otherwise the inferior vena cava is not going to have blood to return to the right heart.

DR. ROBERT P. McBURNEY, Memphis, Tenn.—I certainly agree with Dr. Spencer that in this high velocity age in which we live we are in the future apt to see this type of injury more frequently, and perhaps we have overlooked it in the past.

I would like to show one interesting case. (Slide) This was a 56-year-old man who was in an automobile accident, who had a fracture of the right leg, some broken ribs on the right side, and mild shock. He recovered from shock, and about 3 days later began to complain of some slight dysphagia.

(Slide) This slide shows the lateral roentgenogram, with a mass behind the trachea in the upper mediastinum.

(Slide) A barium swallow done by his physician revealed a marked deviation, and at that time he was referred to me, which was 2 days after the original barium swallow.

(Slide) I think you will note that there is a marked difference between the first barium swallow and the second one, the second one revealing a pronounced increase in the compression

and deviation of the esophagus. When I first saw the patient he was almost completely dysphagic, not even able to swallow liquids.

We made a diagnosis of rupture of the aorta, and elected to operate on him. We used a shunt from the left subclavian artery to the aorta around the area of false aneurysm and rupture.

(Slide) The area of tear was in the classical location, as Dr. Spencer has described. This area was excised and a graft inserted.

(Slide) The patient did well, but a week later when he was walking around, he suddenly went into shock, and it was obvious that something had gone wrong. However, thoracentesis on the left side didn't produce blood, but did yield a large amount of milk which he had drunk a few hours before. A diagnosis of rupture of the esophagus was made, however, the patient's condition would not permit us to reoperate so he was treated by tube drainage and other conservative means. He died, and I would like to show the autopsy specimen.

(Slide) This is one end of the esophagus; here is the other end—in other words, a complete slough and disruption of the esophagus occurred. His death was due to this slough of the esophagus which produced a mediastinal infection, which in turn a week later caused rupture of our suture line, and death was from hemorrhage.

DR. GEORGE MORRIS, Houston, Texas.—We have had an operative experience with 17 patients in the second group with traumatic aneurysm. There have been no deaths in this series. Two of these interesting patients will be shown.

(Slide) This represents a 22-year-old girl who had turned her convertible over 2 years before while in college. The only symptoms were pain. The angioaortogram before operation shows the traumatic aneurysm just distal to the left subclavian artery.

(Slide) The technique we used here was a temporary shunt bypass from the left subclavian artery to the aorta below. This obviates the use of heparin, although in most of these cases we had to use pump bypass.

(Slide) This is the operative photograph, showing the traumatic aneurysm and replacement with Teflon graft. Note the characteristic appearance of the traumatic aneurysm arising in the aortic isthmus.

(Slide) The final result is seen in the angioaortogram showing a normal aortic lumen after resection and replacement with a graft.

(Slide) The second patient had also been in an automobile accident about 10 years previously. He was a 48-year-old man whose doctor had diagnosed his problem as a traumatic rupture of the thoracic aorta, but after seeing the angiogram we were somewhat dubious of the diagnosis, since the aortic contour was quite normal. However, he did, in fact, have a tear in this region with a huge sacciform aneurysm.

(Slide) In this case it was unnecessary to resect any portion of the aorta, and we simply trimmed the area of tear and repaired it with this patch graft. Again we used a shunt from the subclavian artery to the aorta below during the period of cross-clamping. Postoperatively, the angioaortogram was similar to preoperative study, except for the loss of the soft tissue mass.

DR. JAMES R. MALM, New York, N. Y.—A recent review of the literature reveals that of those patients who survive more than 12 hours after the injury, more than 40 per cent of them subsequently die of complications related to their traumatic aneurysm.

We have had experience with only 2 cases of traumatic aneurysms among 126 cases of surgically treated thoracic aneurysms at Presbyterian Hospital, but I would like to present one, since it has an interesting clinical feature that was referred to by Dr. Spencer.

The patient was injured by a car and sustained fractured ribs and a left hemopneumothorax. Following treatment for shock, the blood pressure was 110/70 mm. Hg, but during the first 10 days of hospital he had recurrent chest pains, and blood pressure was recorded in ranges of 150/90 and subsequently 180/100 mm. Hg.

(Slide) The roentgenogram revealed a double contour to the aortic knob, which can be seen in this area and the lateral projection shows the typical location of this lesion.

(Slide) This is not an angiogram. This is a straight lateral projection.

The patient was reluctant to have additional treatment at this time and was followed for a period of 3 months, during which time he had repeated bouts of left ventricular heart failure and ran a brachial hypertension of 210/110 mm. Hg. He was admitted to Presbyterian Hospital in cardiac failure. At that time we noted he had no femoral pulses or abdominal aortic pulsations.

After treatment of cardiac failure, the patient was operated upon through a left posterolateral thoracotomy, and the aneurysm was found just distal to the left subclavian artery. This aneurysm was excised and replaced with a crimped Dacron graft.

(Slide) The last slide will show the pathologic lesion as we saw it. The hematoma had acted as an obturator and simulated coarctation of the aorta. The patient's blood pressure postoperatively was 110/70 mm. Hg. He had good femoral pulses, and he is doing well 2 years later.

DR. SPENCER (Closing).—The case described by Dr. McBurney of esophageal injury is a particularly important one, because the patients reported with this injury have almost uniformly developed a fistula with resulting fatal hemorrhage from the aortic anastomosis. One such case was described in detail by Dr. Shumacker about a year ago.

One reason for our focusing attention upon this lesion is that there is only one case report of successful treatment of traumatic rupture of the aorta in the first day or two after injury (Passaron, E., and Pace, W. G.: Surgery, 45, 787, 1959). We have been unable to find others despite an extensive review of the literature. It is a simple lesion with a transverse tear, and with alertness to the ominous significance of the widened mediastinum I think prompt treatment by the members of this Society can lead to a number of cures in the next year or two.

DR. Å. SENNING, Stockholm, Sweden.—In connection with the two last papers, I would like to show a case that perhaps may be of some interest to you.

It was an 11-year-old boy who was suspected of having a coarctation. One day he had pain in his chest, and in the next few days he developed a high fever and was sent to the hospital. When he came to the hospital he had a hemoptysis of 500 c.c., and a roentgenogram (slide) showed total atelectasis and fluid in the left chest and a bulging of the esophagus, indicating an aneurysm.

We thought this was bleeding, and performed an emergency operation. We found that the patient had a coarctation with a large aortic aneurysm. There was no bleeding in the chest, but there were signs of a mediastinitis. We resected the aneurysm and found that, in the left bronchus, all the posterior membranous wall from the carina down to the lower lobe bronchus was necrotic, and there was also a small foreign body perforation in the esophagus which apparently had given rise to the mediastinitis. We sutured a pericardial flap into the bronchus. We repaired the hole in the esophagus, and sutured down the thymus between the esophagus and the bronchus on one side and the aorta on the other side. We sutured an ordinary De Bakey graft into the resected aorta.

(Slide) The next day there was only a small atelectasis of the left lung (slide), but the roentgenogram of the third day showed complete atelectasis of this lung again. The lung was blown up with a heart catheter in the lower lobe bronchus.

After a few days the patient showed signs of an esophagobronchial fistula. A pharyngostomy for draining was made and a gastric tube was inserted for feeding.

The child recovered without signs of infection. Chest and esophageal roentgenograms were normal as well as aortography. Bronchography showed a completely normal appearance of the bronchial wall in the left main bronchus.

The interesting thing is that, in spite of the fact that he had a mediastinitis, it was possible to do a transplantation of the aorta and the plastic repair of the left bronchus—with the aid of high doses of broad spectrum antibiotics.

DR. O. T. CLAGETT, Rochester, Minn.—Dr. Swenson has called our attention to a source of feeding and respiratory problems in children that has not been understood or recognized adequately in the past.

Dr. Ellis and Dr. Payne have reviewed our experience at the Mayo Clinic with this problem, and in the years 1936 to 1959, 32 patients, 15 years of age and younger, with achalasia were seen. The youngest in which the diagnosis was made was less than a year old. Four were less than 3 years old, and 10 were less than 6 years old at the time of diagnosis, but definite symptoms had existed in 10 of the patients before they reached 1 year of age.

Hydrostatic dilatation of some type had been the treatment of choice in the earlier experience that we are reporting. Twenty-six of these patients were treated by dilatation. Four patients had only one dilatation, but all of them had symptoms again within less than a year. Most of our patients did require multiple dilatations, three or more, and one unfortunate patient had to have 18 dilatations in 15 years in order to keep going.

I think the multiplicity of these dilatations, which averaged about six for this group of patients, attests that dilatation has not been, in our hands at least, a very effective treatment for achalasia in children. It enables them to get by, but it doesn't offer a lasting effect.

More recently, we have operated upon 6 patients with a Heller type of procedure, and in the 4-year follow-up of these 6 patients the results were excellent in 5 and good in 1. The superiority of the Heller type of operation is obvious.

An instructive observation was made in 2 children in whom a preoperative diagnosis of achalasia was made, but who, at the time of operation, proved instead to have a leiomyomatous tumor that was causing the obstruction, and not achalasia.

I am convinced that surgical treatment is better than dilatation. The important thing is to recognize the condition. The attention of pediatricians must be directed to this problem, so that it will be recognized and treated before the esophagus becomes so dilated and so atonic.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—If I interpreted the illustrations correctly, the operative procedure which Dr. Swenson employed to correct the achalasia will shorten the length of the esophagus and may well bring the stomach up above the level of the hiatus opening. It is my firm belief that if such a dislocation of the stomach occurs, the infant or child is a definite candidate for peptic esophagitis and therefore the operative procedure (at least in my hands) is not a satisfactory one. If open surgery is considered, I would be more inclined to perform a myotomy and not disturb the anatomic position of the cardioesophageal junction.

DR. SWENSON (Closing).—I agree with Dr. Clagett that we probably should operate on these patients earlier. However, 4 of the patients with three dilatations each have remained asymptomatic for periods of from 2 to 8 years.

It would seem wise to try two or three dilatations and if these are not effective operation should be undertaken. In performing myotomy we open both layers, simply to secure biopsy material. I think it's interesting that, while we once considered that regurgitation invariably led to peptic esophagitis, we are gradually having to revise this conception. We have seen patients who have had regurgitation for a number of years and have not developed any peptic esophagitis.

DR. DAVID P. BOYD, Boston, Mass.—Of great interest in this splendid paper was the demonstration of a pressure inversion point at which a sharp drop in pH is associated with a sharp drop in pressure. This verifies what we all know but cannot demonstrate anatomically or surgically, namely the presence of a sphincter mechanism at the physiologic cardia. Every fluoroscopist knows of its existence. As a matter of fact, it was beautifully demonstrated a few minutes ago in Dr. Swenson's first movies. Intriguing also was the demonstration of symptoms in relation to peptic activity in the absence of hiatus hernia or obvious regurgitation.

I am sure Dr. Hill does not want this discussion to get into the problem of approach, but he did emphasize that hiatal sutures are not enough, and that has been our experience too. All of us with experience in this field have tried to add steps to make the repair more secure.

Now, in recurrent and complex hernias I have found that the crus of the diaphragm can be very greatly relaxed by separating it from the aorta, and that more relaxation of the diaphragmatic hiatus can be obtained by undermining the pericardium, so that the arms of the hiatus literally fold together, and a few sutures lightly tied are all that is necessary. These steps require a thoracic approach.

In addition, to make the repair secure, fixation of the fascia and fundus can be done from above or below. In large and complex hernias, however, a combined approach has, in my experience, been increasingly useful.

A final word relative to what might be called the undilatable stricture, the last group that Dr. Hill spoke about. We too have had a small but rather gratifying experience with the procedure described by Ellis and Clagett, particularly in patients with very high acidity and duodenal deformity. However, in a few cases in which no duodenal deformity and moderate acidity were evident, good results have also been obtained by high transplantation of the stomach, adding a drainage procedure at the pylorus.

DR. WILLIAM I. WOLFF, New York, N. Y.—Recently we had occasion to review 306 cases of hiatal hernia which were seen over a 10-year period at the St. Vincent's Hospital, the diagnosis being made irrespective of whether or not this was the primary complaint of the patient. We came up with some very interesting findings. We found that approximately two thirds of these patients had symptoms which could be referred to the hiatal hernia.

Of great interest was the fact that 25 per cent of this group, or 1 out of 4 patients, had bleeding in some form, either acute gastrointestinal hemorrhage, severe chronic anemia, or melena, and we took great care to rule out any case in which there was any other demonstrable bleeding of the intestinal tract which might have been responsible.

As Dr. Hill indicated, these patients are often misdiagnosed as having cardiac disorders, particularly in an older patient with dyspnea, because of a severe occult anemia, and substernal pain. Many clinicians have great difficulty in giving up this diagnosis even when an esophageal hiatal hernia is demonstrated.

One out of 7 patients in our group at some time or other had had a gross gastrointestinal hemorrhage; 24 per cent of these patients, again about 1 in 4, have esophagitis demonstrable radiologically or by endoscopic examination. I believe that this esophagitis is of a reflux nature, but I am not entirely convinced that gastric acidity is the determining factor. We, and others, have seen many patients who have had severe esophagitis with either low or no acid in the stomach. Also, we know that some patients have significant reflux for considerable periods with no esophagitis, as Dr. Swenson mentioned.

There was also some indirect evidence. Patients with hiatal hernia who had esophagitis were compared with reference to age with patients with hiatal hernia but without esophagitis. With advancing age the gastric acidity tends to be diminished, and we would expect that the younger age group would tend to have a higher incidence of esophagitis were gastric acidity a critical factor, but such was not the case, and these two curves practically superimpose one upon the other.

The case of persistent esophagitis, even after apparently adequate repair of hiatal hernia, is a problem which has yet to be solved. The speakers gave an interesting demonstration of the proper way to correct this anatomic defect surgically, and I think everything that can be done to prevent persistence of reflux postoperatively will contribute to a good permanent result.

DR. GEORGE C. MORRIS, Houston, Texas.—Our experience demonstrates some changing concepts in this disorder. This slide demonstrates the protean symptomatic

manifestations seen in our patients. The only constant symptom that we have noticed is pain which has been present in 100 per cent of the patients, but this pain is quite varied in character.

(Slide) The operative approach that we have come to use more frequently in recent years is repair through the abdomen. This slide illustrates the duration of hospital stay in repair from above, compared with repair through the abdominal approach, and it shows that hospitalization is considerably shorter with the abdominal approach. More important is the high incidence of associated abdominal lesions which can be handled simultaneously when the abdominal approach is used. In over 50 per cent of our cases there has been some associated abdominal lesion which needed treatment. The most common, of course, is gall bladder disease, but a variety of other associated lesions were also corrected, including abdominal aortic aneurysms.

DR. HILL (Closing).—Those of us who have had the privilege of watching Dr. Boyd use his method of obtaining additional relaxation of the diaphragm know what a valuable aid this is in obtaining enough mobility of the diaphragm to bring it up over some of these large hernias.

DR. ADRIAN LAMBERT, New York, N. Y.—We have been interested in this problem since the early days of intratracheal anesthesia, when some of these patients would be intubated via the transesophageal route!

Recently we have been seeing other cases to which I would like to direct your attention in which there is intrinsic disease of the esophagus, especially that which is due to a corrosive agent.

During a 10-year period on the nose and throat service at Bellevue Hospital under Dr. Daly, 601 patients were admitted with a history of having ingested a corrosive agent. These were all esophagoscoped in order to determine the extent of the burn in the esophagus. Twenty per cent of these patients showed ulcerative esophagitis. The other 484 patients had esophagitis without ulceration. Eighty-five of 97 patients with ulcer who were treated by bouginage by Salzer technique were followed. Of these, 15 developed stricture necessitating gastrostomy and dilatation, and 3 died in the acute phase within the first 2 weeks of treatment.

I would like to show 1 patient who will illustrate this condition, and in whom a perforation occurred at endoscopy. This is a 70-year-old man, an alcoholic, who came in with a 5-day history of having ingested a glass of lye and water, who had been unable to swallow for some 2 days. On physical examination he had edema of the larynx and epiglottis with marked ulceration of the hard and soft palate, with râles in both sides of the chest.

He was esophagoscoped the next day, the esophagoscope being passed down to the cricoid cartilage. He was put on Metacorten, 10 mg. four times a day intramuscularly, with penicillin; during his course he was esophagoscoped four times more, on the fourth time being esophagoscoped down to the 34 cm. point. The scope was withdrawn a little, and a gastric tube was passed with some difficulty, and then passed easily.

That evening the patient after a 300 c.c. meal went into shock. The blood pressure dropped to 80/30 mm. Hg. His temperature rose to 101.4° F. On physical examination it was noted that he had cyanosis of the nail beds. He had rhonchi in his chest with muffled, distant heart sounds. A portable roentgenogram revealed a fluid level, and further x-ray studies revealed the gastric tube to be in the pericardium. He was transferred to the surgical service via the operating room, at which time a pericardiotomy and a superior mediastinotomy were done. It was possible to see the material in the pericardium.

For the next 24 hours his condition was stabilized. He developed marked tracheal secretions with a pulse of 140. The second day his condition rapidly deteriorated, and he died, although he had no further cardiac tamponade.

(Slide) This slide shows the picture of him on admission.

(Slide) The next slide shows him after the tube had been passed, and you can see the air in the pericardium.

(Slide) The lateral view shows the tube in place in the pericardium, which was taken just before his transfer to surgery.

I would like to emphasize that this man was on cortisone, which may mask the precarious condition of these patients to some degree. Our problem is to have these patients brought to surgery before this type of catastrophe has occurred.

DR. LYMAN A. BREWER, III, Los Angeles, Calif.—The authors have made an important contribution in pointing out the dangers of delayed operation in instrumental perforation of the esophagus. In this day and age of antibiotic treatment there is a tendency to cure all infections with antibiotics but here is a situation where relying on the antibiotics may be fatal and prompt surgery is lifesaving. We have had no fatalities in 6 patients in which immediate operation was performed.

The authors have pointed out that this condition is occasionally diagnosed as spontaneous pneumothorax following endoscopy. We have used a method of determining traumatic esophageal pleural fistula that is simple and has been very effective in our hands. The patient swallows a 25 per cent solution of Hypaque (an intravenous contrast medium). This solution is very thin and will flow out through a tiny hole or perforation in the esophagus. Once it enters the pleural cavity, there is a severe pleuritic type of pain which is very characteristic. Thus the diagnosis can be made even before the x-ray film is taken. Using this Hypaque ingestion technique, the diagnosis may be made very quickly after the perforation has occurred.

This early diagnosis of perforation is important, for if there is an organic obstruction it may be possible to relieve the obstruction at the time of the emergency thoracotomy. To my knowledge the concept of carrying out definitive surgery at the time of the treatment of the acute instrumental perforation has not been advocated before in the medical literature. Yet if the diagnosis is made before significant contamination has occurred, then it is logical and safe to go ahead with the relief of the obstruction. The following report is of a case which illustrates that surgical resection of the carcinoma was indicated in this instance immediately after the instrumental perforation of the esophagus.

(Slide) This is an esophogram of a patient with carcinoma of the thoracic esophagus with the characteristic irregular filling deformity in the lower esophagus.

(Slide) Immediately following esophagoscopy the patient experienced severe pain in the right chest. The traumatic right-sided pneumothorax is shown on the chest roentgenogram with shift of the heart to the left side. We were called to see this patient within an hour and the diagnosis of perforation was made. Furthermore, because the endoscopy had been completed, it was obvious from the biopsies that carcinoma was present.

The patient was in excellent shape, so that we elected to go ahead and resect the esophagus because no serious contamination had occurred. This patient had an esophageal resection and high thoracic esophagogastrostomy and Heineke-Mikulicz pyloroplasty. A closed drainage tube was placed in the right pleural cavity. The postoperative course was no different from the usual elective esophagectomy.

The authors have made a very important contribution by calling attention to immediate surgical treatment of the perforated esophagus as the therapy of choice. I would like to recommend carrying the immediate surgical treatment one step further—by completing the definitive surgery whenever an obstructive lesion is present. Certainly this is indicated if the diagnosis is made early, and significant contamination has not taken place.

DR. PAUL C. SAMSON, Lafayette, Calif.—Several years ago Dr. Dugan and I presented our first 29 cases of personal experience with esophageal perforation, and at that time presented also a classification of esophageal trauma (Injuries and Wounds of the Esophagus—A Classification, California Med. 80: 363, 1954). I would now like to add another 20 cases gathered from personal sources, as well as from our services at the VA and County Hospitals.

Now, this series of cases is larger than yours, Dr. Templeton, because we believe that if we are going to talk about perforations, we want to talk about foreign body perforation as well as instrumental.

Surgically the cases can be classified as follows: Operation under 24 hours: 20 cases, Fifteen of these patients were cured without complications, and 5 died. Of those that died, 4 had inoperable carcinomas, and one had a coronary attack. In other words, the closure of the rent was successful in all cases.

Of those operated upon over 24 hours there were 6, 1 cured without complications, 1 cured with severe complications, and 4 died—a 70 per cent mortality.

Those patients whose chests were drained only numbered 9. One was cured without complications, 6 had prolonged complications, and 2 died—22 per cent mortality.

Fourteen patients had no operation; 8 were cured without complications, and 6 died, a mortality of 43 per cent. Of the 8 who were cured without complications, 6 cases were due to foreign body perforations and undoubtedly minute. In the other 2, the surgeon was just lucky.

There are still too many surgeons, internists, and endoscopists who wish to resort to watchful waiting, who think the patients aren't sick enough to be operated upon, or too sick to be operated upon. One need only read some of the recent literature to find this out.

I think we should remember some of our general surgical tenets; namely, that a rent in a hollow viscus ought to be closed, and it seems to me to make little difference whether the esophagus lies in the chest or in the neck; it is still a hollow viscus, and early surgical exploration is highly desirable to say the least.

Any surgeon who does not operate promptly must defend his stand or expect criticism from his colleagues.

DR. THOMAS C. MOORE, Indianapolis, Ind.—I should like to limit my remarks to that phase of this report dealing with the closure of esophageal defects.

Our interest has emanated largely from the experimental laboratory where, for several years, we have been producing relatively large esophageal defects and attempting to close them by a variety of techniques. The most successful technique we have employed was the use of omentum brought into the chest through a hole in the diaphragm. These defects averaged about 2.5 by 3 cm. in length, and our closure success was somewhat in excess of 90 per cent in these animals.

However, if significant contamination of the pleural cavity has occurred, it may subject the patient to additional hazards to open the diaphragm. A recent study completed by us may be pertinent in this regard.

We have attempted to use intact adjacent lung for the closure of these full thickness defects, and have found an incidence of success of approximately 90 per cent. We used two layers of closure. A continuous suture for one of the layers seemed to be slightly more suitable than the interrupted suture technique for both layers. It is quite possible that such additional support will not be required for many of these perforations. However, it may be of some value in questionable closures, or defects that cannot be closed, to remember that the adjacent lung can be employed with success in the closure of full thickness esophageal defects of relatively large size.

DR. RICHARD H. OVERHOLT, Boston, Mass.—It seems that throughout the meeting this week, each discusser has tried to outdo the other in regard to the magnitude of his series. In this particular area I think we ought to compete in the reverse direction. In New England we have not had an opportunity to see many perforations. I have been the author of one myself, and this patient was repaired within about 6 hours.

I vividly remember a patient referred for treatment 15 days after a traumatic rupture, and this case is interesting because of the long interval and the patient's ability to withstand mediastinitis and bilateral empyema for all that time without either medical or surgical supervision.

He was on a hunting trip in the northern part of Maine, and in a drunken wrestling match he found himself on the floor at the bottom of a pile of humanity. Within 2 hours his face and neck began to swell, and his eyes closed.

He slept it off. The next morning the others went hunting, but he could not get out of bed. His face continued to be swollen, and he remained in the hunting camp for a period of 2 weeks, living on sips of whiskey. His friends, of course, thought his difficulty was simply inebriation.

At the end of this time he couldn't walk at all. He was carried out of the woods and taken to an ambulance where oxygen was available. He was taken as far as Portland, Maine, and placed in a hospital.

(Slide) He was almost moribund. There was evidence of bilateral pneumonitis and a massive bilateral air and fluid accumulation was demonstrated by x-ray studies. He was then given, at this hospital, a bismuth swallow (slide) and they were rather alarmed to find that most of the material found its way into the pleural cavities.

He was then transferred to the Deaconess Hospital, and on the same day we drained the left chest and pushed two catheters over into the right chest. No attempt to repair the large opening in the esophagus was made.

A Levin tube was used for feeding purposes, which remained in place for 47 days. He made a satisfactory recovery.

DR. JOSIAH FULLER, Duluth, Minn.—I'd like to rise in support of early operation for instrumental perforation of the esophagus by reporting 6 cases. In response to the last discusser I should say that I didn't split all these esophagi.

These 6 patients ranged in age from 62 to 76. Four of them were over 70. All of them had cervical perforations. Four of these were due to pressure splits. Two of them were actual perforations of the esophagus by the instrument.

All of the patients had early operation. Five of them were able to swallow within 4 days—one of them within 5 days—of their operation. Three of them had early suture. Two of them developed cervical fistula of the esophagus. Cervical perforation at the cricopharyngeus level is the most common instrumental perforation.

I think we should emphasize it's an easy operation. It can be done on a very sick patient, and it can be done as soon as the diagnosis is made, on the basis of fever, pain, subcutaneous emphysema, and findings in the lateral roentgenogram.

A METHOD FOR THE CLOSURE OF SURGICAL PULMONARY INCISIONS WITH ELECTROCOAGULATION

Stuart M. Poticha, M.D., and F. John Lewis, M.D., Chicago, Ill.

THE HISTORY of the use of electric currents in the destruction of living tissue begins with the experiments of D'Arsonval in the latter part of the nineteenth century.[1] He found that the muscular contractions produced by an electric current cease when the frequency of oscillations of that current reaches 10,000 per second, and he defined currents whose oscillations are greater than 10,000 per second as high frequency currents. D'Arsonval also proposed the idea that this high frequency current could be passed through the human body with no other effect than the production of heat.

With these ideas as a foundation, Doyen[2] proceeded to experiment with high frequency currents and their ability to destroy human tissue. In 1907 he presented his findings, describing a method of destroying carcinomatous tissue with an electric current. This was the birth of electrocoagulation.

During the first 30 years of this century, great strides were made in the perfection of electrosurgery. Wyeth[3] and later Bovie[4] invented and perfected new electrosurgical apparatuses. As better and better machines were developed, surgeons incorporated these new techniques into practically every surgical subspecialty. Clark[5, 6] was responsible for clearly defining the methods of fulguration, electrodesiccation, and electrocoagulation. His experiments with electrosurgery were perhaps the greatest single factor in the rapid expansion of these techniques.

By 1935, electrosurgical procedures had been devised for breast surgery, stomach and intestinal resections, hysterectomies, cholecystectomies, and surgery of the thyroid, brain, eye, and skin.[7, 8]

The use of thermic methods in the thorax was initiated by Jacobeus[9] in 1913 with his description of intrapleural pneumolysis. This procedure did not require cauterization of the lung tissue itself, but only of the adhesions between the lung and the chest wall. The cauterization was accomplished with a galvanic current passed through an iron rod.

Graham, in 1925, was the first to use cautery on lung tissue.[10] He found that almost all mortality in lobectomies performed for chronic bronchiectasis was due to septicemia and bacteremia, and reasoned that, if a lobectomy could be performed without opening any vessels and exposing them to infection, this mortality could be appreciably reduced. He devised a method whereby he opened a small window into the thorax and plunged a red hot soldering iron

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into the pulmonary tissue of the infected lung. By repeating this procedure over the ensuing weeks, he eventually destroyed the entire lung, and his results were significantly better than those in which conventional surgical lobectomies were performed.

Since the lung had already been attacked by cautery methods, it is not surprising that it too was caught up in the trend toward electrosurgery. However, only one electrosurgical procedure seemed to evolve about the lung, that is Matson's use of electrocoagulation in performing intrapleural pneumolysis.[11] This procedure was used for several years, but when pneumothorax was generally abandoned in the treatment of tuberculosis there was no longer a need for it.

A careful survey of the literature failed to find any additional pulmonary procedures in which electrosurgical methods were used until 1957. At this time Yakunichev[12] described an experiment contrasting pulmonary resections with the scalpel and with the electroknife. He found only minor changes in the systemic and pulmonary blood pressures with electrosurgery which did not occur with scalpel surgery. The formation of thrombi in the cut vessels was limited to the zone of tissue destruction with both methods. He concluded that the mortality and number of postoperative complications were not increased by using electrocoagulation.

The object of this study has been to investigate the possibility of using electrocautery for segmental and subsegmental lung resections. In particular, we have had in mind the possibility of resecting numerous small diseased portions of lung, as might be required in an operation for extensive bullous emphysema. We thought that electrocoagulation would seal air leaks as well as bleeding points on the raw surface without constricting the remaining functioning lung tissue. Electrocoagulation could be used while the lung was rendered ischemic and airless by clamping its root.[13] This paper is a report of our experiments designed to study this technique.

METHOD

Mongrel dogs, weighing 13 to 19 Kg. were used in these experiments. The animals were anesthetized with pentobarbital sodium, intubated, and attached to a respirator. Using aseptic technique, a thoracotomy was performed in the fourth intercostal space and, after occluding the root of a lobe, a superficial part of the lobe was resected with a knife and scissors. Some of the largest bleeding vessels were tied and then the raw surface was cauterized with electrocoagulation. After the area had been carbonized, the clamps occluding the root were removed and the lobe reinflated. If air leaks appeared in the cauterized area, they were easily sealed with the electrocautery tip. A catheter was brought out through a stab wound but this was only used to re-expand the lung after the thoractomy wound had been closed. It was not left for postoperative drainage. The catheter was removed before the animal was taken off the operating table. Penicillin (100,000 units) was instilled in the chest cavity before closure and the animal was given the same dose daily for 3 days postoperatively.

The surgical Junior Bovie Electrosurgical Unit (Model No. L.H.S. 202-901) was used in this experiment with the unit set for coagulation and power control set at 40 units. The coagulating current produces a maximum damping of the waveform, with a minimum wave train frequency. The power control

Fig. 1.

Fig. 2.

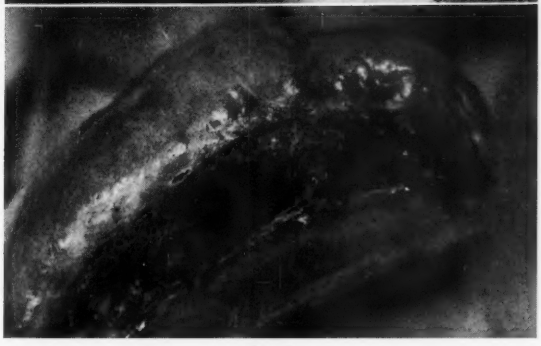

Fig. 1.—Resection of edge of the upper lobe and cauterization of the raw area have been accomplished while the lung root is clamped.
Fig. 2.—The lung shown in Fig. 1 has been reinflated.

is a variometer between two inductance coils and the unit numbers, while arbitrary, correspond roughly to and vary directly with the voltage induced in the coils.

The lung cauterization operations done fall into three series, depending on the site and extent of the cauterization. In Series I (Table I), consisting of 8 operations, two 3 by 1 by 1 cm. strips were removed from the edges of the upper lobe (Figs. 1, 2). In Series II (Table II), a broad (3 by 4 cm.) but superficial area of the left upper lobe was removed and cauterized. In Series III (Table III), the entire costal surface of the right cardiac lobe, which is approximately 5 by 8 cm., was removed (Figs. 3, 4, 5). Series II and III were similar in the nature of the operation but involve different lobes. Series I was a less extensive but deeper type of resection.

Some animals were used for more than one cauterization operation. All of the animals in Series I had two operative procedures and 4 animals in Series II were operated upon twice.

RESULTS

A total of 31 operative procedures was performed in this study, and the healing was observed from 1 to 24 weeks postoperatively. As a general pattern, the cauterized surface became adherent to the parietal pleura or adjacent lung and a fibrous scar developed quite rapidly. The lung deep to the scar appeared to remain functional.

Although these animals were denied postoperative catheter suction drainage and measures to prevent atelectasis, which are always employed after lung resection in man, the incidence of intrapleural and pneumonic complications was not unduly high. One animal in Series I (operation No. 7) died of pneumonia 6 days after its second lung resection. One animal in Series II (No. 10) and one in Series III (No. 31) died of empyema. A tension pneumothorax caused the death of 1 animal in Series II (No. 13) and atelectasis caused the death of an animal in Series III (No. 26). Of the other deaths, one was an anesthetic death (No. 12), and in animals Nos. 8, 9, 17, and 28 which died 4, 7, 9, and 12 days postoperatively there was no apparent cause of death. In each of these, the lung appeared to be breathing and it had reached the stage of healing noted in the animals sacrificed at similar periods postoperatively.

On gross inspection of the healing lung in Series I, in which the surface resection was less extensive, the scar was narrow and the remaining lung tissue appeared to be functioning normally. The lobe in Series II and III in which surface cauterization was extensive appeared to have its original functional volume reduced by one third to one half due to scar formations. In one dog in Series III (No. 24), the remaining lobe appeared to be nonfunctional.

In all three series, the healing process observed microscopically was much the same. The cut ends of the blood vessels were occluded immediately by the current. Ellis[14] has described the process by which these vessels occluded. The muscular layer of the wall of the arterioles and venules undergoes contraction and fusion, thereby obliterating the vessel lumen. In addition, some endothelial lining cells break off and plug the vessel. Kuntzen and Vogel[15] showed that actual thrombus formation played only a small part in the closure of these vessels.

Microscopic sections at 1 week showed the periphery of the lesion attached to the parietal pleura. Immediately deep to this was hematoma and necrotic tissue. This tissue presents the appearance of the hyalinization which has been described by Bierman.[14] The architecture of both the tissue and individual cells is completely destroyed, and one homogenous amorphous mass is left without any signs of cell membranes, nucleus, or cytoplasm. Underlying this was a thin layer of fibrous tissue which covered the lung. The lung tissue showed an intense inflammatory reaction in the area immediately adjacent to the scar (Fig. 6).

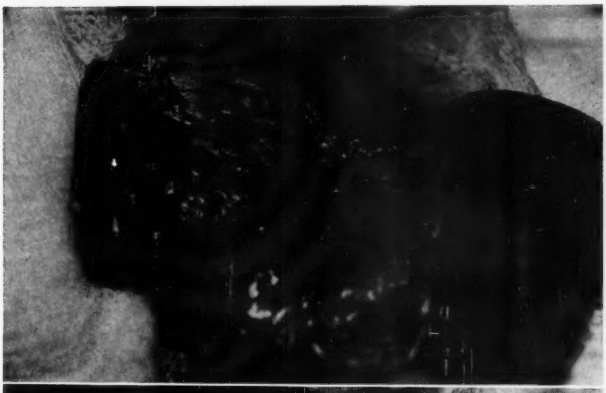

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 3.—Right cardiac lobe untreated.
Fig. 4.—Right cardiac lobe with root clamped and surface excised.
Fig. 5.—Right cardiac lobe after cut surface has been cauterized.

TABLE I. SERIES I.—RESECTION OF THE EDGE OF THE UPPER LOBE

OPERATION NUMBER	NUMBER OF POSTOP. DAYS	REMARKS
1	7	Lung well healed
2	14	Lung well healed
3	21	Lung well healed
4	6	Lung well healed
5	28	Lung well healed
6	170	Lung well healed
7	6	Died after the second resection; pneumonic infiltration in operated lobe
8	7	Died after second resection; lung well healed; no cause of death

TABLE II. SERIES II.—SURFACE RESECTION OF UPPER LOBE

OPERATION NUMBER	NUMBER OF POSTOP. DAYS	REMARKS
9	4	Died; lung well healed; no definite cause of death
10	8	Died; necrosis of entire left lung with a massive empyema in the left hemithorax
11	7	Lung was well healed with 50% of lobe functioning
12	0	Anesthesia death
13	12	Died due to air leaks and tension pneumothorax
14	21	Lung was well healed with 70% of lobe functioning
15	28	Lung was well healed with 70% of lobe functioning
16	14	Lung was well healed with 70% of lobe functioning
17	9	Died; no definite cause of death established; 50% of the lobe involved in scar
18	157	Lung was well healed with 70% of lobe functioning normally
19	144	Lung was well healed with 70% of lobe functioning normally

TABLE III. SERIES III.—SURFACE RESECTION OF THE RIGHT CARDIAC LOBE

OPERATION NUMBER	NUMBER OF POSTOP. DAYS	REMARKS
20	42	Lung was well healed with 60% of lobe functioning normally
21	45	Lung was well healed with 70% of lobe functioning normally
22	42	Lung was well healed with 60% of lobe functioning normally
23	12	Died; no definite cause of death; lung was well healed with 30% of lobe involved in scar
24	7	The wounded lobe was atelectatic
25	43	Lung was well healed with 70% of lobe functioning normally
26	7	Died; the entire right lung was atelectatic
27	97	Lung was well healed with 70% of lobe functioning normally
28	97	Lung was well healed with 70% of lobe functioning normally
29	95	Lung was well healed with 60% of lobe functioning normally
30	7	Lung was well healed with 70% of lobe functioning normally
31	4	Died; necrosis of the right middle lobe with empyema of the right hemithorax

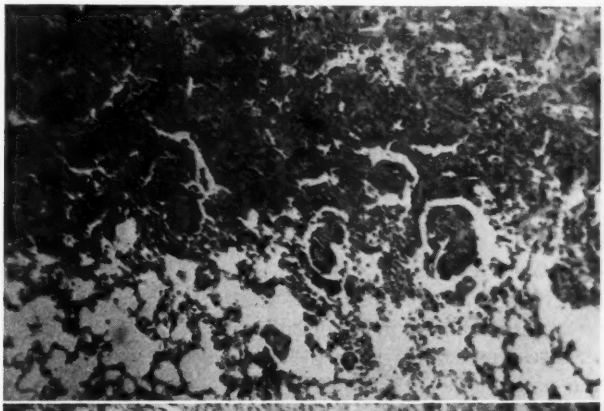

Fig. 6.

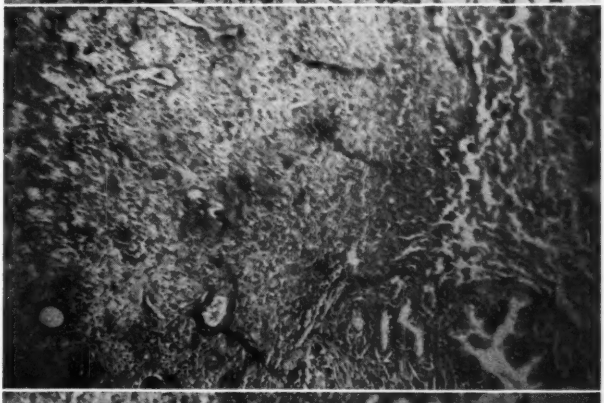

Fig. 7.

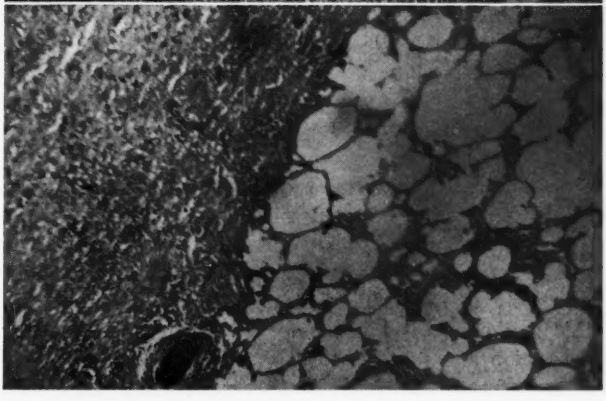

Fig. 8.

Fig. 6.—Microscopic section at 1 week which shows necrotic hyalinized tissue overlying inflamed lung.

Fig. 7.—Microscopic section at 2 weeks which shows fibrous tissue proliferation throughout scar. Lung tissue still inflamed. Scar is at the left.

Fig. 8.—Microscopic section at 3 weeks which shows thick fibrous scar overlying normal lung tissue.

At two weeks the picture remained much the same; however, early fibrous tissue proliferation was seen entering the hematoma and hyalinized tissue, both superficially from the parietal pleura and deep from the fibrous scars overlying the lung (Fig. 7).

The process then consisted of the replacement of this amorphous tissue by proliferating fibrous tissue both superficially and deep. The inflammatory reaction in the underlying lung tissue gradually disappeared in the ensuing weeks.

The final result at 3 weeks showed a thick firm fibrous tissue scar which was attached to the parietal pleura and chest wall superficially and underlain by normal lung tissue (Fig. 8).

In Series III in which a portion of the right cardiac lobe was resected, this area became adherent to the right upper lobe. The healing process was similar, the visceral pleura covering the upper lobe contributing in the same manner as the parietal pleura.

DISCUSSION

These experiments demonstrate that electrocautery is an effective method of sealing off blood and air loss from the raw surface of the lung. Although large surface wounds were made, they healed satisfactorily and without a high complication rate. The infrequency of difficulty due to air leak postoperatively was striking, for no chest drainage was employed. There was definite evidence of an inadequate airtight seal in only 1 case (No. 13). The fact that this animal died on the twelfth postoperative day leads one to believe that there was inadequate adhesion formation or a premature separation of the eschar. In either case this could, of course, be a serious consequence and its occurrence must be kept in mind in contemplating this procedure on human patients.

An important feature of this technique is vascular and bronchial occlusion of the lung root. With this occlusion the resection and subsequent cauterization are easily carried out in a bloodless field. Guided by some earlier studies,[13] we began by occluding the pulmonary artery, veins, and bronchus individually. It was thought that bleeding from the bronchial arteries would pose a serious problem in maintaining sufficient hemostasis so that the resected area could be cauterized adequately. By occluding the bronchus individually with a Satinsky clamp, it was possible to compress the bronchial vessels against the cartilage of the bronchus and thereby prevent any bleeding from this source. However, in later experiments it was found that the bronchial circulation was not troublesome and that it was unnecessary to occlude these arteries. In Series II and III, all the major structures in the hilus were occluded simultaneously with one clamp and this method proved adequate for controlling hemostasis and air leaks during resection and cauterization.

The ultimate effect of the type of current used in these experiments is maximum heating of the tissue. The higher the voltage, the more quickly the tissue is dehydrated. By using a comparatively high voltage (power control at 40 units), the tissues immediately adjacent to the active electrode are quickly dehydrated. This carbonized tissue then acts as an insulator, preventing the

deeper tissues from being heated and damaged. A much higher voltage would destroy only the most superficial tissue and such a current would not form an airtight seal over the cut bronchioles. A much lower voltage would heat the tissue slowly thereby devitalizing a much greater amount of normal lung. These experiments showed that with the power control set at 40 units a compromise was reached in that the eschar formed was deep enough to occlude the bronchioles while destroying only a minimum of normal lung tissue.

We hoped that this method of treating the raw surface after partial resection would result in less constriction and limitation of the remaining lung than conventional methods with sutures. This is a difficult thing to measure and our experimental studies do not settle the question. It is evident, however, that in most cases the lung scarring due to electrocautery did not appear to be excessive. We think that this technique may improve segmental and subsegmental resections and that its trial in man is justified. The technique obviously cannot be used with explosive anesthetic agents.

SUMMARY

1. A technique of partial lung resection employing occlusion of the vessels and bronchi with electrocoagulation of the raw surface was studied in 31 dog operations.

2. Despite minimal postoperative care and no intrapleural suction drainage, the lung wound usually healed satisfactorily. There was surprisingly little trouble with air leaks in the postoperative period.

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SECOND PRIMARY BRONCHOGENIC CARCINOMA

A Complication of Successful Lung Cancer Surgery

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IN A RECENT ARTICLE Burford, Center, Ferguson, and Spjut[1] have analyzed the results of surgical therapy at Barnes Hospital for bronchogenic carcinoma. The 5-year survival curve for the 182 patients with resectable lesions falls rapidly during the first 30 postoperative months but thereafter reaches a gently downward sloping plateau. The authors found that the 63 patients surviving at least 30 months had a 75 per cent chance of becoming 5-year survivors.

In following the postoperative course of these 63 patients, an unexpected finding was encountered—4 of them (6.4 per cent of the 30-month survivors) developed a second primary bronchogenic carcinoma. Having experienced successful surgical therapy for one primary lung cancer, these patients survived for a sufficient period for another primary lung cancer to become manifest. In other words, from the standpoint of cancer therapy, the second primary cancer may be legitimately regarded as a complication of successful lung cancer surgery.

Although there have been other reports of multiple primary lung cancers,[2-5] this series places emphasis on those cases in which a second pulmonary malignancy, separated from the first by a long asymptomatic interval, presents the clinical and pathologic findings of a new primary lesion rather than a recurrence or a metastasis. The objective of this paper is to report these cases and to discuss their significance. The findings in the 4 cases of second primary bronchogenic cancer are summarized in Fig. 1.

CASE REPORTS

CASE 1.—C. G. B., age 47, was admitted to Barnes Hospital in May, 1953, for complaints which had started in October, 1952, when the patient experienced increasing shortness of breath, and a productive cough with occasional blood-tinged sputa. A chest film taken 1 month prior to admission was reported to show a "spot" in the right lung.

The patient had smoked cigarettes for 30 years; two packages per day for the last 15 years. The patient's father had died of cancer of the rectum and his mother had died of cancer of the uterus.

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No significant physical findings were noted on admission and bronchoscopy was also unrevealing. Chest films, however, showed a radiopacity in the pectoral segment of the right upper lobe and bronchial washings from the right side were found to contain cancer cells. Two subsequent sputum examinations were also positive.

On May 15, 1953, a right thoracotomy was performed and the right lung was resected. Grossly the lung (53-2755) was well aerated and the pleura free of adhesions. A 2 cm. hard mass was found obstructing the lumen and destroying the walls of the pectoral

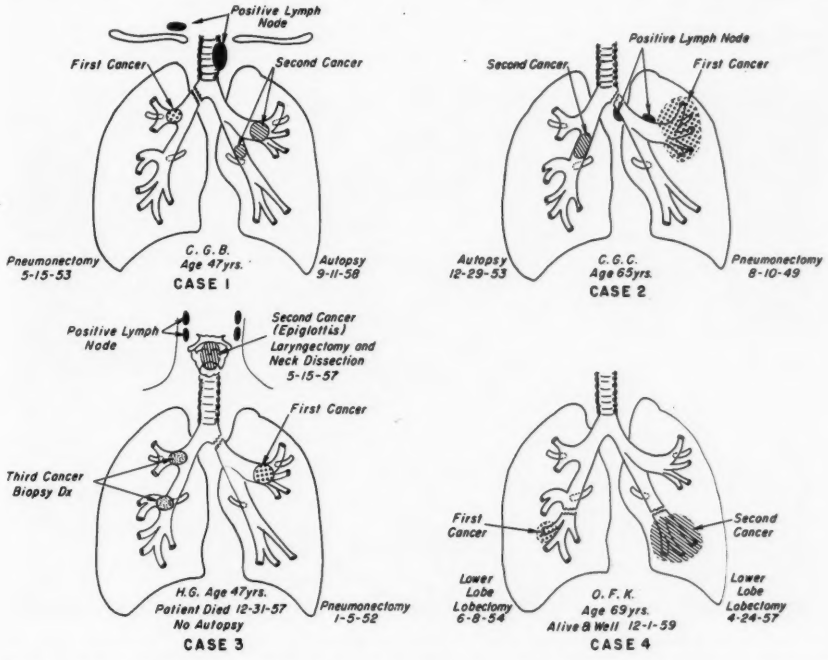

Fig. 1.—Schematic representation of the 4 cases of second primary bronchogenic carcinoma (W.U. Ill. No. 59-7644).

and subapical segments of the upper lobe bronchus, with extension into an adjacent bronchopulmonary lymph node (Fig. 2). Histologically the tumor was a well-differentiated squamous cell carcinoma with frequent areas of incomplete keratinization. The tumor projected into the lumen of the bronchi, invaded the surrounding lung and adjacent pleura and metastasized to one paratracheal and three anterior superior mediastinal lymph nodes. The line of resection of the bronchus was free of tumor and there was no vascular invasion or secondary nodules. Seven tracheobronchial lymph nodes were free of metastases. Recovery was uneventful and the patient was discharged on May 25, 1953.

The appearance of blood-tinged sputa prompted a readmission to the hospital in February, 1954. Chest films showed no significant changes. No recurrent tumor could be seen on bronchoscopic examination. The cytologic examination of the bronchial washings reported them as being suspicious but malignant cells could not be found on two subsequent sputum examinations.

The patient was followed closely and was doing well until the onset of progressive hoarseness necessitated readmission to the hospital in August, 1957. Laryngoscopy demonstrated a paralyzed left vocal cord and marked deviation of the trachea to the right. No

tumor recurrence was detected on bronchoscopy. A left scalene node biopsy (57-6162) showed a number of normal lymph nodes on microscopic examination. Cytologic examination failed to show any cells on the smear of the sputum.

In January, 1958, a chest film showed marked deviation and angulation of the trachea to the right and emphysema of the left lung. Because of increasing shortness of breath, a right thoracoplasty was performed. The patient had difficulty with secretions during the postoperative period because of the vocal cord paralysis but he recovered satisfactorily with slight respiratory improvement.

However, the patient's respiratory difficulties progressively increased until the final admission on Sept. 9, 1958, when he also complained of left chest and scapular pain. The clinical impression was recurrent tumor in the anterior mediastinum and the patient was

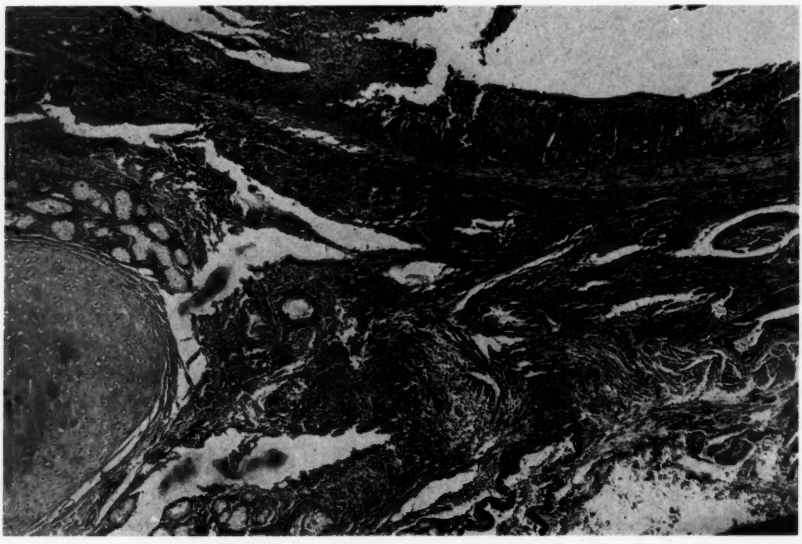

Fig. 2.—Endobronchial epidermoid carcinoma with contiguous carcinoma-in-situ and with invasion of the submucosa is seen in the right upper lobe bronchus (W.U. Ill. No. 59-7627). (×50; reduced ⅛.)

given two courses of nitrogen mustard therapy (10 mg.). Subsequently the patient required constant endotracheal suction to clear the respiratory tree of secretions and the patient died 2 days later of respiratory failure. The patient had abstained from smoking since the pneumonectomy.

At autopsy (19255), the right pleural cavity was obliterated by scar tissue without evidence of cancer. The stump of the right main-stem bronchus and the right pulmonary artery were without recurrence of cancer. A few fibrous adhesions were present in the left pleural cavity but no fluid was encountered. The upper and lower lobe bronchi of the left lung (850 grams) were involved by two separate cancers, each less than 1 cm. in diameter (Fig. 3, *A* and *B*). Both were moderately well-differentiated squamous cell carcinomas. Both grew within the lumen of the bronchi and spread into the surrounding lung tissue (Figs. 4, 5, 6).

The metastases in the lymph nodes had a very unusual distribution. The hilar and left tracheobronchial lymph nodes were uninvolved but the mediastinal, right paratracheal, and left supraclavicular lymph nodes were destroyed by tumor. A mixed histologic pattern was found in the superior mediastinal lymph nodes; some areas showed the incomplete keratinizing pattern seen in the pneumonectomy specimen while adjacent areas resembled those seen in the autopsy specimen. No visceral metastases were found.

A. *B.*

Fig. 3.—A granular tumor is seen growing into the lumen of the left upper lobe bronchus *(A)*, and the left lower lobe bronchus (arrow) *(B)*. (W.U. Ill. No. 58-5946 and No. 58-5944.)

Fig. 4.—The cross section of the left lower lobe bronchus shows predominant intraluminal tumor growth at the top of the picture and parenchymal invasion below. The apparent discontinuity of the cancer in the photograph is a result of the plane of section. Fig. 5 is taken from the enclosed area in this picture (W.U. Ill. No. 58-5945).

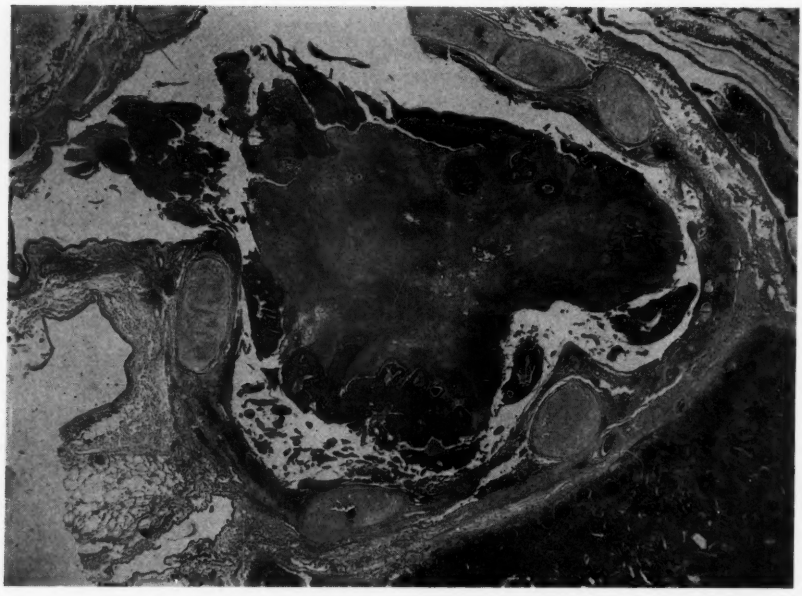

Fig. 5.—A low-power photomicrograph showing the lumen of the left lower lobe bronchus occluded by cancer (W.U. Ill. No. 59-7624). (×8; reduced ⅛.)

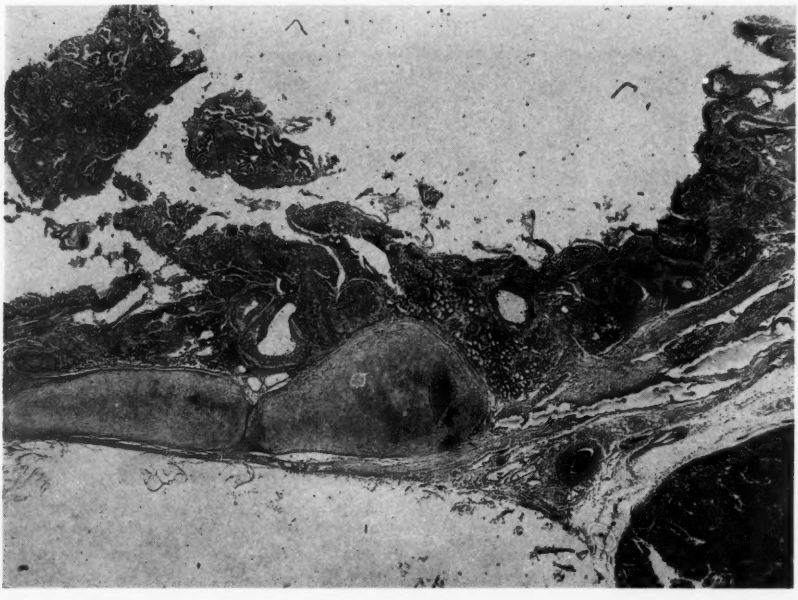

Fig. 6.—A similar view of the left upper lobe bronchus is seen (W.U. Ill. No. 59-7621). (×12; reduced ⅛.)

CASE 2.—C. J. C. was admitted to Barnes Hospital for the first time in August, 1949, at the age of 64, with complaints of a chronic cough recently associated with left anterior chest pain and weight loss.

Physical examination showed signs of consolidation and fluid in the left lung while chest films and fluoroscopy demonstrated a well-demarcated, homogeneous mass in the left upper lung field. Central cavitation of the mass with an air-fluid level could be seen on some films. No significant roentgenographic changes were seen in the right lung fields or hilar regions.

Bronchoscopy showed a moderate amount of mucoid secretion draining from the left upper lobe bronchial orifice and a biopsy (67564) from this region showed only squamous metaplasia. Cytologic examination of the secretions, however, demonstrated the presence of malignant cells as did a subsequent sputum sample.

The left chest was explored on Aug. 10, 1949, and the left lung was resected. On gross examination of the specimen (67661), a large (8 by 10 cm.) tumor with central necrosis and cavitation was found occupying the subapical segment of the left upper lobe and extending across the interlobar fissure to invade the apical portion of the lower lobe. Bronchi to the apical division of the left lower lobe were compressed and displaced from their normal position. Microscopically the tumor proved to be a poorly differentiated epidermoid carcinoma with vast areas of necrosis. No vein invasion was demonstrated and the bronchus at the line of resection was free of tumor. One of 10 tracheobronchial nodes and one of 14 bronchopulmonary nodes contained metastases.

The patient experienced an uneventful recovery from the operation and was discharged on Aug. 19, 1949. He was readmitted 2 months later with a bronchopleural fistula and a left empyema which were treated surgically by open drainage. Two sputum examinations at this time were negative for malignant cells. Chest x-ray studies during a third Barnes Hospital admission in 1950 for malum coxa senilis showed no evidence of disease in the remaining lung. The patient did fairly well until the fall of 1953 when he experienced increasing shortness of breath, weakness, recurrent weight loss, and right anterior chest pain; he was admitted to the hospital on Dec. 28, 1953, severely dyspneic and cyanotic, and died 24 hours later.

At autopsy (16099), the left pleural cavity was partially obliterated by dense scar tissue and a shifted mediastinum. No gross or microscopic tumor could be found in the scar tissue or in the stump of the left main bronchus or pulmonary artery. The right pleural cavity contained 1 L. of transudate and the right lung (2,000 grams) showed complete consolidation. A carcinoma, 4 by 6 cm. in size, was found astride the lower lobe bronchus and impinging upon the other main bronchi. Chronic suppurative pneumonitis with abscesses was found in all lobes. Histologically the tumor was a poorly differentiated squamous cell carcinoma with rare foci of keratinization. At some levels it was largely endobronchial in extent but at other levels the obliteration of the bronchus was complete with invasion of the adjacent lung tissue. No lymph node or visceral metastases were found.

CASE 3.—H. G., a 47-year-old white male truck driver, first entered Barnes Hospital on Jan. 2, 1952, complaining of pain in his left chest, blood-streaked sputum, and cough of 6 weeks' duration. He had smoked two packages of cigarettes per day for 15 years. Physical examination showed dullness and râles over the left upper lung field. Radiographs were interpreted as showing atelectasis and pneumonitis of the left upper lobe. Bronchoscopic examination showed no lesion; however, the left upper lobe orifice was not well visualized. Cytologic examination of the sputum and bronchial washings demonstrated cancer cells. On Jan. 5, 1952, a left pneumonectomy was performed (52-76). A 1.5 by 1 by 1 cm., well-differentiated epidermoid carcinoma was found that arose at the junction of the apical posterior and anterior bronchi of the left upper lobe and partially obstructed the lumens. The distal lung parenchyma was involved by organizing pneumonia. The line of resection was free of tumor. Thirty bronchopulmonary and mediastinal lymph nodes were free of metastatic tumor. The postoperative course was uneventful and he was discharged on Jan. 16, 1952.

Five and one-half years later, June 16, 1957, he returned complaining of a mass in the right side of his neck that had been present for 2 years with slow increase in size. He had returned to work, continued to smoke as before, and had been in good health. Biopsy of the neck mass (57-4655) proved it to be metastatic epidermoid carcinoma. The primary lesion (41058) was found to be at the base of the epiglottis and a transhyoid subtotal laryngectomy with bilateral radical neck dissection (41121) was done. Four upper cervical nodes of a total of 82 nodes, two on each side of the neck, contained metastatic epidermoid carcinoma. He recovered, had some difficulty learning to swallow, and was discharged.

Five months later, Nov. 9, 1957, he returned with complaints of nausea, vomiting, and weight loss. No evidence of persistent carcinoma was seen in the larynx, hypopharynx, or neck. Radiograms showed a right hilar shadow. Bronchoscopy demonstrated a granular lesion involving the right upper and middle lobe orifices. Biopsy (42017) of this proved it to be undifferentiated carcinoma. The patient received 2,518 r tumor dose over 14 days via the betatron and was discharged without symptomatic or radiographic improvement. He died at home on Dec. 31, 1957. No autopsy was performed.

CASE 4.—O. F. K., age 69, was admitted to Barnes Hospital on May 28, 1954, for the evaluation of a lung opacity found on a routine chest examination. The patient had smoked a pipe and cigarettes for many years but was asymptomatic on admission. At the time, he was under therapy for Laennec's cirrhosis, proved by needle biopsy. Chest x-ray studies demonstrated a lesion in the posterior basilar segment of the right lower lobe which was interpreted as carcinoma of the lung and a metastatic lesion in the lingular division of the left upper lobe was also suggested. Bronchoscopy was negative and bronchial washings showed only a few atypical cells. The patient underwent a right exploratory thoracotomy on June 8, 1954, and a mass was felt in the basilar medial segment of the right lower lobe. The mediastinum appeared to be free of tumor. A right lower lobectomy was performed.

A firm, gray-white tumor, measuring 4 cm. in diameter, was found involving the anterior superior basal bronchus (54-3371). Microscopically the tumor was a moderately well-differentiated epidermoid carcinoma with extensive necrosis. No vein invasion or lymph node metastasis was recognized grossly or microscopically. The patient recovered uneventfully and was discharged on June 17, 1954.

The patient was without respiratory complaints until February, 1957, when he developed a persistent cough with blood-tinged sputum following an upper respiratory infection. Bronchoscopy in March, 1957, showed no evidence of recurrent tumor. An irregular, lobulated infiltrate in the left lower lobe with an air-fluid level was seen on chest roentgenograms and was interpreted as demonstrating cavitating carcinoma. Cytologic examination of the bronchial washing was negative. On April 24, 1957, a left thoracotomy was performed and the lower lobe of the left lung was removed, after a frozen section confirmed the presence of cancer. The pleura was thickened and retracted over a 5 cm. centrally placed mass, 4 cm. from the line of bronchial resection (57-3085). Central cavitation within the mass was present grossly. Two small segmental bronchi were found directly involved by tumor. No other masses were found. Microscopically the tumor was an epidermoid carcinoma with the same degree of differentiation as the previous lung cancer. A remnant of bronchial epithelium was found adjacent to tumor in the center of the mass. No lymph nodes were found to be involved with metastases and no epithelial changes were seen at the line of bronchial resection. The patient recovered without difficulty. When last seen in December, 1959, he was in good health without evidence of disease and had adequate respiratory function.

DISCUSSION

The subject of multiple primary cancers has been argued for more than a century and the conclusions are often conflicting. One of the major difficulties has been the choice of criteria for deciding if and when a second lesion

is truly a second primary cancer. Some authors have found the criteria of Billroth, as cited by Warren and Gates, excessively stringent and have considered those of Warren and Gates[5] more reasonable and useful. These latter criteria which have been applied in our cases, stipulate that, "each of the tumors must present a definite picture of malignancy, each must be distinct, and the probability of one being the metastases of the other must be excluded." From the preceding case histories it should be obvious that the first two stipulations are adequately met, and we will consider briefly the various possibilities for metastatic spread of lung cancer to satisfy the third stipulation.

In our cases the possibility of the second lesion arising from direct extension from the first one is remote. All of the lines of resection of the initial surgical specimens were found free of tumor. Furthermore, the bronchial stumps of the autopsied cases (Cases 1 and 2) and the resected ends of the bronchi in the second surgical specimen (Case 4) showed an absence of carcinoma on careful examination. In no case was there invasion of the lower mediastinum, pericardial sac, or widespread lymphatic dissemination. Cases 3 and 4 had no lymph node metastases in the surgical specimens and in Case 2 no involved pulmonary or mediastinal lymph nodes were found at postmortem. Metastases to the upper mediastinal and paratracheal lymph nodes were found at the initial surgery in Case 1. At autopsy, however, no tumor was evident in the hilar or left tracheobronchial lymph nodes. It is difficult to attribute the origin of the second primary lesion in this case to lymphatic spread without involvement of these lymph node groups.

Hematogenous spread of lung carcinoma is a common and well-known finding, but hematogenous spread to the opposite lung is relatively infrequent.[6] The most frequent sites of metastases are liver, bones, adrenal, kidney, and brain and, when present, they are seldom solitary. But none of these organs contained metastatic deposits in the autopsied cases (Cases 1 and 2). It is true that two widely separated lesions were found in the opposite lung in Case 1 but, in the absence of other metastases, they are not interpreted as secondary metastases but rather as multicentric cancers of about the same stage of evolution.

Intrabronchial spread and implantation have been suggested as routes of spread of lung cancer,[7, 8] but no extensive submucosal spread was encountered in our cases and implantation is seriously considered only in the rare instances of papillomatous tumors of the larynx and tracheobronchial tree that have a totally dissimilar natural history and histologic picture.

The foregoing arguments are largely negative in character and, as such, exclude the possibility of a metastatic origin of the second lesion rather than include the possibility of a new primary. Histologic considerations are of some importance in 2 of our cases. In Cases 1 and 3 the first lesion was a well-differentiated epidermoid carcinoma whereas the second lesion on biopsy showed either an undifferentiated carcinoma (Case 3) or an epidermoid carcinoma with scant keratinization (Case 1). It even seems that one of the anterior superior mediastinal lymph nodes was involved by both cancers as

both histologic types were present in the same node (Case 1). Relatively little weight should be given to these observations as the propensity of lung cancer to show varying histologic patterns in different areas of the primary and its metastases is widely recognized.

Much more convincing is the finding in 2 cases of a tumor with both intraluminal and parenchymal growth and with transitional mucosal changes at the margins.

An important consideration in our cases arises from the clinical fact that the second lesion followed the first by a period of about 33 months. According to Warren and Gates,[5] "the average duration from the onset of those earlier (first primary lesion) tumors to death in our cases (of multiple primary cancers) is three years." This information, coupled with the observation of Burford, Center, Ferguson, and Spjut, that patients surviving at least 30 months have a 75 per cent chance of becoming 5-year survivors, makes it seem unlikely that the second lesion in our 4 cases is actually a delayed recrudescence from the original carcinoma. The second cancers made themselves evident at a time when the first cancer would have been lethal without surgery and at a time when chances for survival were good because of successful surgical intervention. Putting all of these considerations together it seems to us that these 4 cases do indeed represent second primary bronchogenic carcinomas and should be considered as a complication of survival resulting from successful lung cancer surgery. The term "second primary carcinoma" has already been used by Howard and Williams,[2] in 1957, in describing a case of bilateral, simultaneous, primary, squamous cell carcinoma of the lung.

The discovery of these cases of second primary carcinomas among the survivors of lung cancer surgery should not have come as a surprise, in fact, it even could have been anticipated. In the recent past many pathologists, such as Black and Ackerman,[9] Auerbach,[3] Williams,[10] and Ryan and McDonald[11] have shown that bronchial carcinoma is frequently associated with the atypical mucosal alterations and with carcinoma-in-situ. These changes are found adjacent to the invasive cancer, as in 2 of our cases (Cases 1 and 2), or at some distance from it or even in the opposite lung. The occurrence of a secondary cancer in the remaining respiratory tree should not be surprising in view of these findings.[12]

A direct parallel may be found in carcinoma of the oral cavity, another site where carcinoma-in-situ and invasive cancer frequently coexist.[13-15] In the experience at the Ellis Fischel State Cancer Hospital, 8 instances of second primary carcinomas developing after successful treatment of the initial tumor were found in 206 patients with oral cavity carcinoma.[15] One reasonable view is that the second primary cancer appears as a consequence of surgical survival that enables in-situ cancer of the bronchial mucosa to become invasive and have clinical manifestations of its own. A striking example of a susceptible respiratory tract to carcinomatous changes is found in Case 3. This patient survived a primary carcinoma of the lung and larynx, both with lymph node metastases, only to succumb to a second carcinoma of the opposite lung.

This raises an interesting point: any highly successful therapy for lung carcinoma may not only have to obliterate the initial cancer but also have to deal in some way with a respiratory tract that is cancer prone. No such therapy suggests itself at present, but the problem is not insignificant. The 4 cases reported here represent 6.4 per cent of the 63 patients surviving 30 months or more.

The second cancer was a major factor in causing the respiratory failure responsible for death in Cases 1, 2, and 3. Hence it is possible to attribute 20 per cent (3/15) of the deaths that occurred during the third, fourth, and fifth postoperative year of survival to the second primary carcinoma. And these percentages should be regarded as minimum values because not all deaths during this time period were examined by autopsy. The figures obtained, however, provide ample reason for careful and repeated follow-up studies on all patients surviving lung cancer surgery in excess of 30 months.

It is of interest that the 3 patients who died had a pneumonectomy as the initial surgical procedure whereas the 1 survivor (Case 4) had a lobectomy. Fortunately, the second primary lesion was also resectable by means of a lobectomy and the patient remains alive to date. This suggests that a lobectomy is preferable to a pneumonectomy whenever the choice can be made without impairing the initial chances for cure.

Cytologic examinations of sputum were not done routinely in these 4 cases. In spite of the fact that the few isolated examinations failed to detect the second primary cancer, periodic cytologic examinations of sputum may well prove helpful in following 3-year survivors for the early detection of second primary carcinoma. It may be especially valuable in particular cases in which recurrent pulmonary symptoms exist and radiographic and bronchoscopic findings are absent or equivocal.

SUMMARY

In following the postoperative course of 63 30-month survivors of lung cancer surgery, 4 cases (6.4 per cent) of second primary bronchogenic carcinoma were discovered. An evaluation of the clinical histories and pathologic findings, together with pertinent facts on patient survival after pulmonary cancer surgery, provides acceptable reasons for considering these patients as having second primary lung cancers. Three of the patients, all having undergone pneumonectomy as initial therapy, died of respiratory failure shortly after the discovery of the second pulmonary malignancy. In these patients the second cancer was discovered during the fifty-second, sixty-fourth, and seventy-first postoperative month of survival. They represent 20 per cent of the 15 survivors who died during the third, fourth, and fifth years of survival. One patient, who had a lobectomy as the initial procedure and who developed a second contralateral primary lesion after 33 months, is alive and free of disease 32 months after a second lobectomy.

The study provides ample justification for careful and repeated follow-up examinations for all patients surviving lung cancer surgery in excess of 30

months. The return of pulmonary symptoms, a new radiographic shadow, or a positive cytologic report in these particular patients should not be taken as prima facie evidence of recurrence, but rather as the first possible manifestation of a new primary cancer. The successful outcome of the patient who survived two primary cancers, each treated by lobectomy, suggests that lobectomy is to be preferred over pneumonectomy in those cases in which the choice between the two exists without impairing the chances for initial cure. Routine sputum cytologic examinations may prove of value in the early detection of a second primary cancer, especially in those cases with return of unexplained pulmonary symptoms.

From the standpoint of cancer therapy, the development of a second primary bronchogenic cancer may legitimately be considered as a complication of successful lung cancer surgery as we know it today. In view of the apparent parallel with oral cavity cancer and in recognition of the association of carcinoma-in-situ and invasive carcinoma in widely separated regions of the respiratory tree the development of second primary carcinomas of the lung should come as no surprise and could have been anticipated. Any highly successful therapy for lung cancer may not only have to obliterate the manifest cancer but also will have to deal in some way with a respiratory mucosa that is cancer prone.

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THE SURGICAL TREATMENT OF SUPRAVALVULAR AORTIC STENOSIS

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PARTIAL OBSTRUCTION to the flow of blood in its passage from the left ventricle to the aorta may result from stenosis of the aortic valve or of the outflow tract (subvalvular zone) of the left ventricle. Either of these obstructions may be congenital or acquired. Supravalvular aortic stenosis, which is a congenital lesion, has been far less frequently encountered.

Two types of congenital lesions have been encountered in the supravalvular portion of the aorta. The most common of these is a fibrous band or cord crossing or subtending the lumen of the aorta just above the cusps of the valve. Ten such examples are recorded,[1-7] and in the majority of these the band extended between the commissure of the left and noncoronary cusps to the commissure between the left and right cusps. Such a band apparently does not cause aortic stenosis or cardiac disability.

The other congenital supravalvular aortic lesion is the type representing the subject of this report, namely, supravalvular aortic stenosis. Four examples have been reported from necropsy material. Kreel and associates[8] wrote that the first report of an example of this lesion was made by Mencarelli[9] in 1930. An illustration of such a condition was found by Denie and Verheugt in Lev's book.[10] Cheu and associates[11] reported upon a 33-year-old patient who had a crescent-shaped fibrous membrane that encircled three fourths of the circumference of the lumen of the aorta just above the aortic cusps. Burry[12] noted constriction of the aorta just at the valvular opening in a 37-year-old patient, with valvelike ridges forming further narrowing at this point. In addition, Sissman and associates[13] discovered, at necropsy, mild supravalvular aortic stenosis in a 70-year-old woman in whom the lesion had not been hemodynamically significant.

Although only four instances of supravalvular aortic stenosis had been noted until 1958, reports have now appeared of 2 cases in which this condition was encountered unexpectedly among patients operated upon for aortic stenosis.

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Denie and Verheugt[14] operated upon a 25-year-old man for congenital aortic stenosis on Sept. 10, 1957. They encountered "a distinct circular narrowing of the aorta immediately distal to the valvular ring," and identified this supravalvular location of the stenosis with the aid of appropriate pressure tracings. No attempt was made to repair the lesion. The patient died at the termination of the procedure as a result of hypotension and irreversible ventricular fibrillation.

Kreel and his associates[8] reported a case in which a 12-year-old boy was operated upon for congenital aortic stenosis on Feb. 25, 1958, with the use of extracorporeal circulation to permit direct observation of the lesion. Through an aortotomy, a slight annular constriction of the aorta was noted immediately distal to the cusps of the aortic valve. "This stenosis was reduced by repeated manual dilatation." The right coronary cusp seemed to be absent. Aortic insufficiency was noted after perfusion, and this resulted in the death of the patient before completion of the operation. At postmortem examination the presence of the supravalvular stenosis was confirmed, and the right coronary cusp was found to be closely adherent to the aortic wall, thus covering the orifice of the right coronary artery.

Morrow and associates[15] recently described 3 patients who had supravalvular aortic stenosis, in 2 of whom the diagnosis was established preoperatively by means of left-heart catheterization and selective angiography. Two of the patients died, 1 after surgery and 1 of cardiac failure. In the third patient, no attempt at correction of the lesion was made at the time of thoracotomy.

Three patients who had congenital aortic stenosis localized to the supravalvular area have been encountered by the present authors. This condition was an unexpected finding in the first and third patients, but the correct location of the stenosis was anticipated in the second patient on the basis of preoperative studies conducted by one of us (Vlad). The technique of repair was identical at each operation and each patient obtained a satisfactory result.

SURGICAL TECHNIQUE

The scheme of surgical approach in operations upon patients who have supravalvular aortic stenosis is the same as that employed for the commoner forms of aortic stenosis. Exposure is obtained through a median sternotomy. The standard system of whole-body perfusion is employed, with cannulation of the right external iliac or femoral artery and the venae cavae via the right auricular appendage.

After the institution of whole-body perfusion, the ascending aorta is cross-clamped just proximal to the origin of the innominate artery. Immediately thereafter a short incision is made in the exposed surface of the ascending aorta about 1 or 2 cm. proximal to the aortic clamp. Through this incision the intracardiac sucker is introduced into the aorta. As soon as visualization is adequate the incision is carried caudally, in a direction spiraling somewhat to the right and posteriorly, so that the lower limit of the incision points toward

the mid-portion of the noncoronary aortic sinus. The nature of the aortic stenosis is then carefully assessed by inspection and by palpation of the supravalvular, valvular, and subvalvular areas. When it has been determined that the typical ringlike supravalvular constriction is present, the aortotomy is carried directly through this ring and perpendicularly to it, well down into the noncoronary aortic sinus and almost to the point at which the base of the corresponding semilunar cusp attaches to the wall of the left ventricular outflow tract. It is especially important thus to carry this incision well downward and entirely through the stenotic zone. A prosthesis of compressed

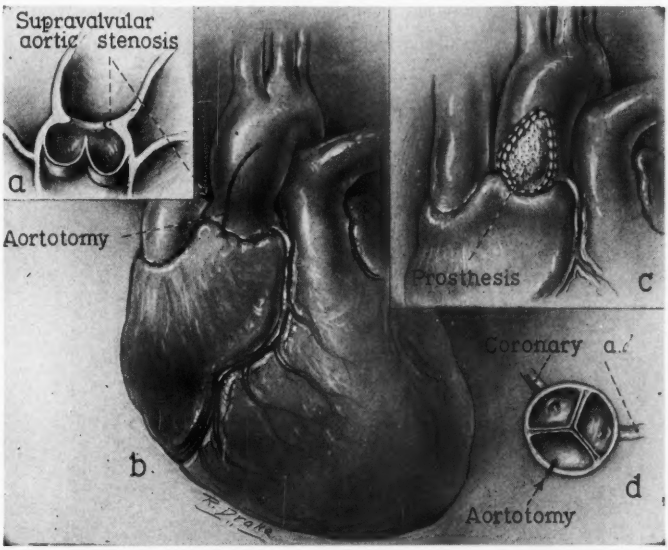

Fig. 1.—Supravalvular aortic stenosis and its repair. *a*, Longitudinal section of aorta, demonstrating the level of the stenosis. The internal ridge causes a more severe stenosis than is apparent externally. *b*, Spiral course of the aortotomy to the right and posteriorly. *c*, An Ivalon prosthesis is sutured to the edges of the aortotomy, effectively increasing the diameter of the aorta at the level of the stenosis. *d*, The aortotomy is carried far down into the noncoronary aortic sinus.

(approximately 4 to 1) polyvinyl (Ivalon) sponge or a plastic cloth material, such as woven Teflon, which is nearly impervious to heparinized blood, is then fashioned in an appropriate diamond shape. The length of this prosthesis equals that of the aortotomy, and it should be so constructed that its widest portion, which is 2 to 3 cm. in width, will come to lie in the same plane as the narrowest portion of the aorta. It is sutured into the aortotomy, as is illustrated in Fig. 1, with continuous 3-0 arterial silk sutures.

We have made it a practice to institute coronary arterial perfusion when the aorta is to be cross-clamped for longer than 20 or thirty minutes. A flow of 400 to 500 c.c. of blood per minute perfuses the coronary arteries through modified Kay coronary cannulas. Coronary arterial perfusion was employed only in the second patient of this series.

In all three instances in which this technique was employed the aortic valve remained totally competent after the corrective procedure, and cardiac action was adequate and excellent thereafter. Extracorporeal circulation was gradually discontinued in the usual manner, and appropriate measurements of pressure suggested the relief of the aortic stenosis.

CASE REPORTS

CASE 1.—An 11-year-old boy, who had been known to have a heart murmur since birth, began in April, 1956, to experience episodes of substernal and subxiphoid thoracic pain both nocturnally and on exertion. This pain was not sustained beyond a few minutes. The relief obtained with nitroglycerin seemed genuine. He had no episodes suggestive of syncope or left ventricular failure.

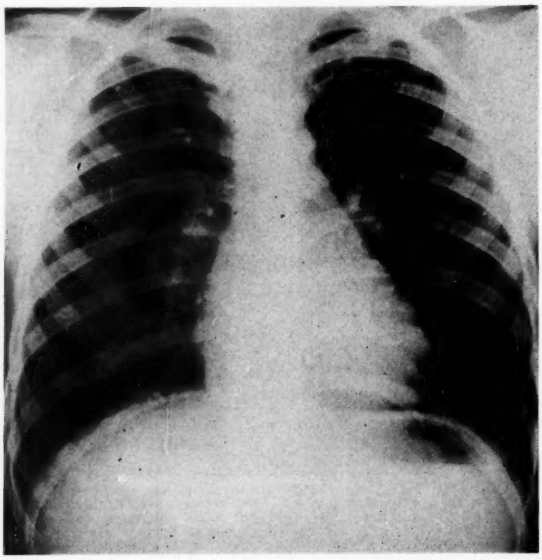

Fig. 2.—Case 1. Preoperative standard roentgenogram of the thorax of the 11-year-old patient. See text for interpretation.

The boy appeared well. A systolic thrill was palpable over the manubrial area and neck. A rough Grade 3 (on the basis 1 to 4) stenotic-type systolic murmur in the aortic area was well transmitted into the neck, and only mildly toward the apex. The basilar second sound was split and of normal intensity. The femoral pulses did not feel full. The blood pressure was 118 systolic, 80 diastolic in millimeters of mercury.

The roentgenogram of the thorax was not remarkable, the size of the heart being at the upper limits of normal, and the vascularity of the lungs was not unusual (Fig. 2). The electrocardiogram showed complete left bundle branch block, with exaggerated P waves present in standard Lead II. Review of an electrocardiogram made early in 1956 showed left ventricular hypertrophy with strain and no evidence of the intraventricular conduction disturbance. It was assumed that the left bundle branch block, which appeared some 2 months before operation, was the end result of left ventricular strain rather than being a postinfarction type of block. This interpretation is suggested by the disappearance of the pattern of left bundle branch block from the electrocardiogram by the fourth postoperative day (Fig. 3).

A diagnosis of aortic stenosis was established on clinical grounds. Operation was performed on Aug. 2, 1956, by the techniques described, except that bilateral anterior thoracotomy was employed. Cardiac arrest was instituted by injection with potassium citrate. After the heart was exposed, the systolic pressure in the left ventricle was 200 mm. Hg, while that in the femoral artery was 82 mm. Hg. After the aorta had been opened, a tight ring of aortic narrowing was identified just above the ostia of the coronary arteries at the level at which the commissures attached to the aorta itself. The orifice through this narrowing was estimated to be not more than 3 mm. in diameter. The aortic valve itself appeared normal. After the repair, the left ventricular systolic pressure was 86 mm. Hg and that in the femoral artery was 63 mm. Hg.

The recovery from operation was uneventful. At re-examination 10 months later the patient seemed asymptomatic. A Grade 2 systolic murmur was still present over the

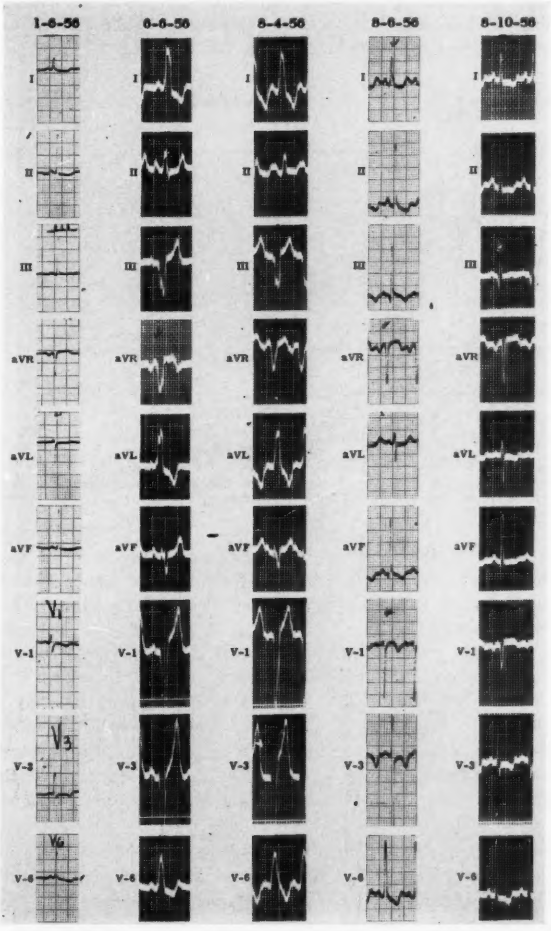

Fig. 3.—Case 1. Electrocardiograms made on the dates indicated above each set of tracings. The first two panels are preoperative curves showing appearance of the complete left bundle branch block. The last three panels are postoperative curves showing the disappearance of the complete left bundle branch block 4 days after operation; this situation had persisted to the time of this writing.

aortic area. The left bundle branch block remained absent, but the electrocardiogram continued to show left ventricular hypertrophy with a strain pattern (Fig. 3). The blood pressure was 122 systolic and 74 diastolic, in millimeters of mercury. A recent report from the patient's home cardiologist would indicate no further change in the boy's condition, save for rather vague thoracic discomfort which is occasionally experienced and may represent angina pectoris.

CASE 2.—A 15-year-old high school girl in whom a heart murmur was first discovered at the age of 7 years had been entirely asymptomatic and normally active throughout her life.

A loud, coarse aortic "ejection-type" murmur was audible over the entire precordium and neck, but was maximal over the manubrium, where a systolic thrill also was palpable. The basilar second heart sound was present. The right carotid pulse seemed full and rather bounding, with a coarse thrill over it, but these signs were much less striking over the left carotid artery. Similarly, the left radial pulse was much weaker than the right. The cuff blood-pressure determinations in the arms were curiously difficult to determine, but the systolic pressures seemed to be about 110 mm. Hg bilaterally.

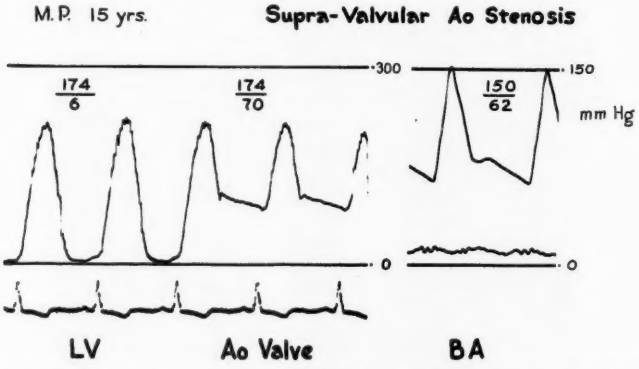

Fig. 4.—Case 2. Pressure tracing made during withdrawal of the tip of the catheter from the left ventricle through the aortic valve into the substenotic zone of the aorta, showing no systolic gradient across the valve. The simultaneous brachial arterial pressure is of course distal to the stenosis.

The roentgenogram of the thorax showed slight enlargement of a somewhat globular heart. The aortic arch was not apparent in the posteroanterior view. The electrocardiogram showed striking evidence of left ventricular hypertrophy and possible myocardial ischemia, with T-wave inversions across the entire precordium. Special studies* consisted of retrograde catheterization of the left heart, with the taking of appropriate measurements of pressure and injection of contrast medium through the same catheter to delineate the aorta. The results of these studies are illustrated in Figs. 4 and 5, and conclusively demonstrate the supravalvular location of the stenosis, which, prior to these studies, had not been appreciated.

Although asymptomatic, the patient was advised to undergo surgical treatment of the aortic stenosis because of the evidence of significantly increased work of the left ventricle. The operation was accepted and was performed on May 14, 1959. The method of repair was exactly the same as has just been described.

At operation, a coarse systolic thrill was palpable over the ascending aorta, innominate artery, and left carotid artery, but the orifice of the left subclavian artery seemed stenotic, and almost no pulse could be felt in the vessel beyond. A definite narrowing of the aorta, just above the aortic sinuses, was apparent. When the aorta was opened, this constricted ring of aorta was seen to lie at the same level as that at which the leaflets of the aortic valve

*These studies were carried out by one of us (Dr. Vlad) in cooperation with Dr. E. C. Lambert, at Children's Hospital, Buffalo, New York.

were attached to the wall of the aorta at their commissures. The lumen of the aorta at that level was estimated to be 6 or 7 mm. in diameter. The aortic wall seemed thickened in this area, forming an inwardly protruding ridge which contributed to the stenosis. The aortic valve itself seemed normal. Prior to repair, the left ventricular systolic pressure was 190 mm. Hg, the blood pressure in the arm, obtained with the sphygmomanometer, being 140 mm. Hg. After the repair, the left ventricular and arm systolic pressures were identical at 140 mm. Hg.

The recovery from surgery was uneventful. A softer, short, coarse systolic murmur was still present over the manubrium, and strong pulsations of the carotids were noted. The blood pressure in the right arm was 110 mm. systolic and 60 mm. diastolic, but only a systolic reading of 84 mm. Hg could be identified on the left.

The patient became normally active and asymptomatic again, and a definite reduction in the size of the heart was apparent 2 months after the operation.

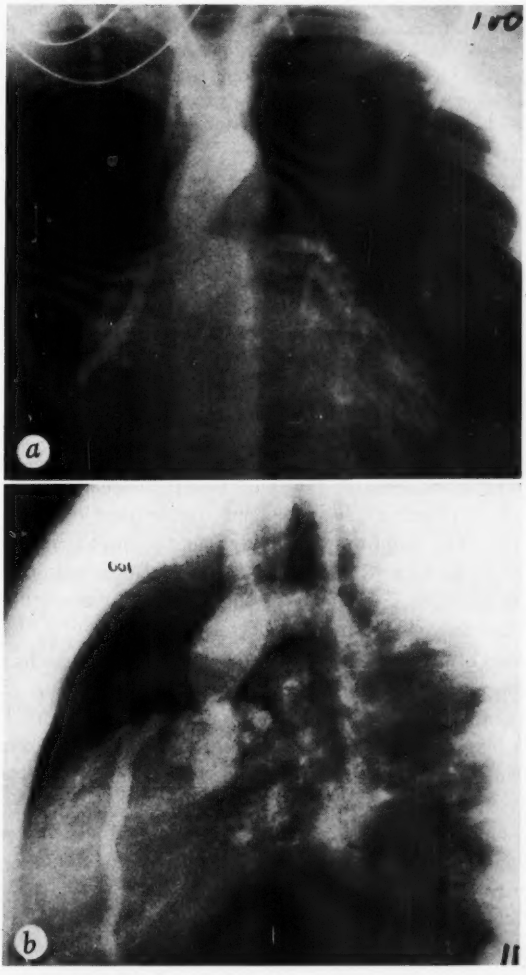

Fig. 5.—Case 2. Retrograde aortogram, demonstrating the supravalvular stenosis in posteroanterior *(a)* and left anterior oblique views *(b)*. The coronary arteries are demonstrated to originate proximal to the stenosis.

CASE 3.—A 14-year-old boy had no symptoms, but had been known to have a heart murmur since the age of 3 years, and his activities had been somewhat restricted by his physician since the age of 11 years.

The boy was large and normal, except for the cardiovascular system. There was a systolic thrill in the suprasternal notch and aortic area. A harsh, rough, Grade 4 (on the basis of 1 to 4) systolic murmur was heard in the right first interspace and was transmitted into the neck and upper part of the back. It was also heard over the entire precordium. Diastole was clear and no ejection "click" was audible. The second aortic sound was obscured. The femoral pulse seemed normal.

A roentgenogram of the thorax disclosed nothing significant. The electrocardiogram showed left ventricular overload of the systolic type, and indicated an ischemic pattern of left ventricular strain and suggested left atrial enlargement.

A diagnosis of severe aortic stenosis was made. At operation on Nov. 14, 1959, the root of the aorta appeared to be normal externally, but when it was opened typical supravalvular stenosis was encountered. A fibrous ring, encircling the interior of the aorta at the level at which the aortic leaflets attached to the aortic wall, had constricted the aortic lumen at this point to a diameter of 6 mm. The aortic leaflets were normal.

It appeared impossible to resect the fibrous ring. Consequently, the stenosis was repaired by the technique described, with the use of woven Teflon for the prosthesis. The pressure in the left ventricle remained at 135 mm. Hg before and after the repair, but the flow of blood at the final pressure reading presumably was much increased, since the aortic pressure was only 85 to 90 mm. Hg before repair and was 115 mm. Hg thereafter.

The patient's recovery was uneventful. A Grade 1 to 2, slightly rough, aortic systolic murmur remained at the time of this writing.

COMMENT

In the 3 patients operated upon by us the supravalvular stenosis was not accompanied by valvular deformity, the occurrence of which would have greatly complicated the repair. It is possible, however, that if a cusp were adherent along its free margin to the aortic wall, as in the case of Kreel and associates, it could be sufficiently freed by sharp dissection to become normally functional. If not, prosthetic replacement or revision of such a cusp would become necessary. The technique of enlarging the supravalvular stenotic ring in these patients is similar to the technique sometimes employed for relief of pulmonary stenosis, when a hypoplastic right ventricular outflow tract or pulmonary artery and valve ring are present.

When unusual clinical findings are made in a patient who has aortic stenosis, such as in our second patient, in whom the pulses in the left side of the neck and in the left arm were much weaker than those on the right, left-heart catheterization and selective angiography are indicated. Thus, an unusually located stenosis of the aortic arch could be identified and hence a proper surgical approach could be made. In the absence of any clinical clues of an atypical aortic stenosis, such special studies may not be routinely necessary, since the surgical approach is currently identical whether the stenosis is subvalvular, valvular, or supravalvular.

SUMMARY

Supravalvular aortic stenosis has been encountered rarely. Three patients who had this condition have been operated upon at the Mayo Clinic, with relief

of the stenosis and survival of the patients, by use of the technique described. The stenotic supravalvular ring was first divided and then permanently enlarged by the suturing of an appropriate diamond-shaped prosthesis in the aortic incision. Whole-body perfusion was employed to provide the conditions necessary for the performance of the procedure. The diagnosis of this condition can be made by the technique of retrograde arteriocatheterization.

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SUPRAVALVULAR AORTIC STENOSIS: DIAGNOSIS AND TREATMENT

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CONSIDERATION of the morbid anatomy, diagnostic methods, and surgical techniques for the correction of supravalvular aortic stenosis assumes increasing importance as surgery for aortic valvular and subvalvular stenosis becomes more commonplace. A review of a patient with supravalvular aortic stenosis, diagnosed by left heart angiocardiography and surgically corrected, forms the basis of this report.[1, 2, 12, 13, 15]

CASE REPORT

R. W. (D.H. 26-47-73), an 8-year-old white girl, was admitted to Doernbecher Hospital of the University of Oregon Medical School in December, 1959, for cardiac surgery. A heart murmur had been discovered at the age of 6 months. The past history and family history were unremarkable. There were no symptoms of cardiac failure, syncope, dizziness, or cyanosis. Mild exercise intolerance had been noted during the year prior to admission. Physical examination revealed a blood pressure of 98/72 mm. Hg in the right upper extremity and normal peripheral pulses. There was no cyanosis or clubbing. A systolic thrill was palpable over the upper sternal area and a Grade 4-6 systolic ejection murmur was audible over the precordium, loudest in the second right intercostal space and transmitted to the neck. Liver and spleen were not palpable and the extremities were normal. The electrocardiogram suggested left ventricular hypertrophy, and chest x-ray studies (Fig. 1) revealed normal heart size with a concavity of the right upper cardiac border which suggested a narrow ascending aorta. Left heart catheterization was performed by the introduction of a polyethylene catheter over a spring guide through the right brachial artery. Selective angiocardiography with the injection of contrast medium above and below the aortic valve demonstrated obstruction to left ventricular outflow just above the level of origin of the coronary arteries (Fig. 2). The aortic valve leaflets appeared normal and the left ventricular wall was thickened.

Operation was performed (A. S.) on Dec. 9, 1959. The heart was approached through a midline sternotomy incision. The supravalvular stenosis was easily visible externally (Fig. 3). The valve ring was of normal size and there was post-stenotic dilatation of the ascending aorta above the narrow segment. A systolic thrill was palpable in this area. The left ventricle was enlarged. Pressure tracings from the left ventricle and femoral artery were made prior to and following correction (Fig. 4). The patient was placed on cardiopulmonary bypass at a flow of 2.6 L. per square meter of body surface area per minute and a satisfactory perfusion was apparent. The apex of the heart was lifted from its bed and a catheter was placed in the left ventricle for decompression. An oval patch of

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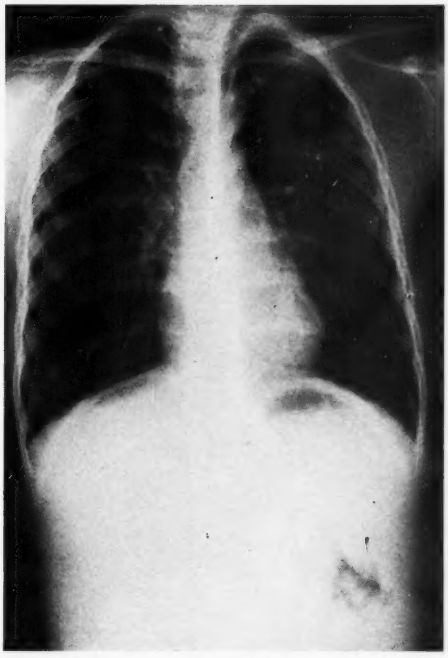

Fig. 1.—The preoperative chest roentgenogram reveals normal heart size. Evidence of widening of the ascending aorta is absent. On the contrary, the right supracardiac border is less prominent than normal.

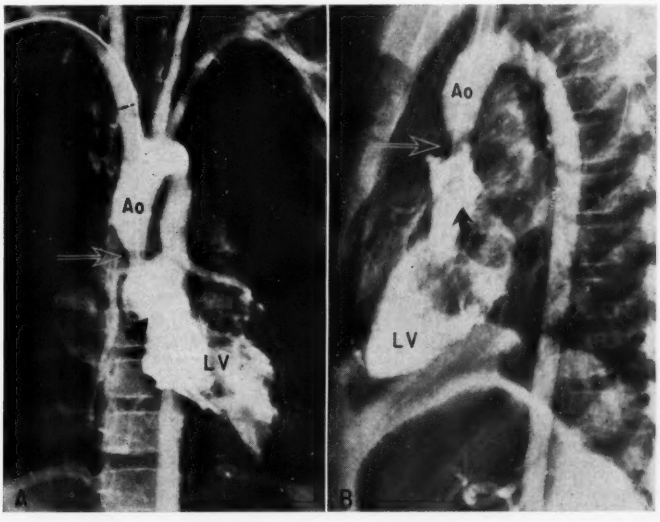

Fig. 2.—Left heart visualization following the injection of contrast substance into the left ventricular cavity. Spring guided catheterization via the right brachial artery route was employed. *A*, Frontal view. *B*, Left lateral view. Both views clearly demonstrate the stenosis as evidenced by a localized supravalvular aortic narrowing with an associated transverse radiolucency due to the circumferential nature of the lesion. The relationship of the narrowed zone to the coronary arteries and the more proximal aortic sinuses is clearly demonstrated. The aortic cusps are well shown above the black arrow on *B*.

Fig. 3.—This photograph of the operative field prior to correction reveals the site and extent of the supravalvular stenosis. The aorta is dilated above the stenosis.

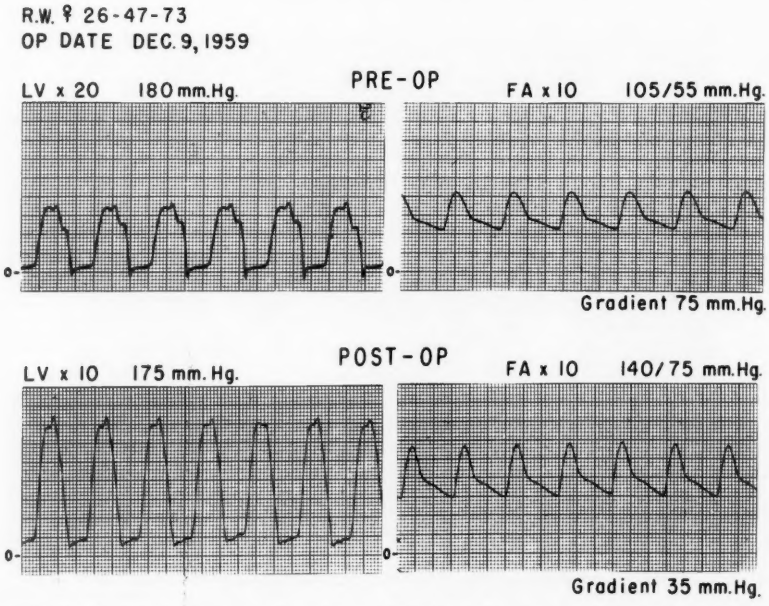

Fig. 4.—Pressure tracings performed at the time of operation immediately before and after correction. The left ventricular to femoral artery gradient is shown. Following correction, femoral artery pressure was increased and left ventricular pressure was slightly reduced.

woven Teflon cloth, measuring 4 by 3 cm., was prepared. The aorta was cross-clamped distal to the narrowed segment and an aortotomy was performed, beginning 5 cm. above the aortic valve ring and extending downward into the sinus of Valsalva of the noncoronary cusp. The distal aortic wall appeared normal but, as the area of stenosis was transected, it was found to be unusually tough and fibrous. The aortic wall of the noncoronary sinus below this narrowed segment was of normal thickness. The aortic valve itself was found to be normal and the stenosis was just above the level of the origin of both coronary arteries. The internal surface of the aorta was markedly thickened at this point by a ring of fibrous tissue. Because the

Fig. 5.—This photograph shows the Teflon patch sutured in place in the longitudinal aortotomy which was made through the stenotic area into the sinus of Valsalva of the noncoronary cusp. The right atrium is retracted downward to expose this area.

aortic valve commissures were contiguous with this fibrous ring, no attempt at resection was made. The previously prepared patch was sutured into the aortotomy with continuous 3-0 silk sutures. The aortic clamp was removed following 11 minutes of myocardial ischemia and a sinus rhythm returned immediately. Oozing from the suture line was well controlled by a vest of compressed polyvinyl plastic sponge temporarily sutured over the Teflon patch. Following administration of protamine, the polyvinyl vest was removed and no bleeding was evident.

Decannulation was effected without incident and the wound was closed in the routine manner. The appearance following repair is shown in Fig. 5. The patient was returned to the recovery room in excellent condition with normal arterial and venous pressures, fully awake, and weighing 325 grams heavier than her preoperative weight. The postoperative course was benign and she was discharged on the fifteenth postoperative day. At the time of discharge, physical examination revealed normal pulsations in all extremities. There was no thrill and there was a Grade 2 systolic ejection murmur audible over the second interspace to the right of the sternum and transmitted to the neck. Electrocardiogram showed left bundle branch block which had been present from the time of surgery.

DISCUSSION

Obstructing lesions in the supravalvular area of the aorta are a heterogeneous group of congenital anomalies. A consideration of 17 cases of autopsy reported in the literature suggest the division of these lesions into three distinct groups.

The first group includes those patients in whom there is a fibrous band with free edges extending across the aortic lumen at the level of attachment of the aortic valve commissures to the aorta.[4, 9] The band usually extends from one commissure of the left cusp to the other and may be associated with atrophy of the left cusp resulting in aortic insufficiency.[17, 18] Most often this lesion is encountered as an incidental autopsy finding and its clinical significance is obscure.

The second group includes those lesions characterized by an obstructing membrane inserting into the aortic wall at the level of the commissures. Cheu and associates[6] have described a patient with this anomaly in whom aortic insufficiency resulted from fusion of the left aortic cusp with this membrane.

A third group includes those lesions in which an externally apparent concentric narrowing or coarctation of the aorta just above the level of the coronary ostia is the dominant feature. In this anomaly there is significant obstruction to flow and clinical heart disease. Thickening of the plica of the sinuses of Valsalva forms an encircling ridge of fibrous tissue. In some cases the free margin of one or more aortic valve leaflets is adherent to the aortic wall, resulting in aortic insufficiency. In rare cases, this may compromise filling of a coronary artery by partially closing the associated sinus of Valsalva. On microscopic examination, the stenosing ridge is shown to be composed of collagenous tissue and elastic fibers continuous with the aortic media. The intima over the ridge is thickened and the adjacent media occasionally reveals fibrosis and cystic degeneration. The valve leaflets show variable thickening due to fibrosis.[5, 8-11, 14]

Clinically, lesions included in the third group are indistinguishable from congenital valvular stenosis. As in the case presented in this report, there is a loud systolic ejection murmur heard over the entire precordium, maximal over the aortic area, and transmitted to the neck. The electrocardiogram shows left ventricular hypertrophy. Chest x-ray examination adds little to differential diagnosis, although there may be a suggestive narrowing along the right supracardiac border. Pressure tracings obtained by percutaneous puncture of the left ventricle are of no value in localizing the stenosis.[3] A pull-out tracing following retrograde catheterization of the left ventricle via the right brachial artery is of some diagnostic value but in one reported instance was not correctly interpreted preoperatively.[8] Diagnosis is best made by left heart angiocardiogram with injection of contrast material directly into the left ventricle and ascending aorta through a catheter placed in retrograde fashion from the femoral or brachial arteries. This study is routinely used in all cases of aortic valvular disease prior to surgery in this clinic. The presence of associated aortic insufficiency is easily detected by a reflex of the contrast agent into the left ventricle following

supravalvular injection. Thus, the more complex anomalies involving adhesions of the aortic leaflets to the supravalvular ridge that result in aortic insufficiency may be diagnosed preoperatively.

Surgical Technique.—Kreel and associates[10] have reported a patient who was operated upon with a diagnosis of aortic stenosis and insufficiency. Using cardiopulmonary bypass, manual dilatation of the supravalvular stenosis was performed. The heart could not be resuscitated because of aortic insufficiency resulting from adhesions of the right cusp to the supravalvular ridge. Morrow and associates[14] have reported an operative procedure on a patient with supravalvular aortic stenosis and dissecting aneurysm of the ascending aorta diagnosed preoperatively by left heart angiocardiogram. A gradient of 60 mm. Hg was present across the stenosis, and aortic insufficiency was not evident clinically. Using cardiopulmonary bypass and elective cardiac arrest, the aortic root was opened and the lumen of the aorta at the site of constriction was enlarged by means of a diamond-shaped prosthesis of compressed polyvinyl sponge. After 67 minutes of cardiac arrest, coronary perfusion was restored but without producing an effective heartbeat. Autopsy examination revealed that the aortic leaflets were free of adhesions to the stenosing ridge. Failure of resuscitation was most likely related to the prolonged period of cardiac arrest.

The patient reported in this article was fortunate in that the aortic valve itself and the aorta beyond the stenosis were normal. The policy of performing left heart angiocardiogram routinely prior to aortic valve surgery led to a precise preoperative diagnosis that was totally unexpected. Since it was known preoperatively that the stenosis was immediately above the level of the sinuses of Valsalva the initial aortotomy incision extended down into the noncoronary sinus. The stenotic area was widened by a woven Teflon prosthesis with 11 minutes of aortic cross-clamping.

Woven Teflon was chosen as the material of choice rather than compressed polyvinyl sponge because the latter has not proved adequate in the past for aortic replacement.[7] Compressed polyvinyl sponge is of uncertain tensile strength and may dilate or rupture when subjected to systemic blood pressures. In addition, woven Teflon cloth has been used in this clinic for widening the right ventricular outflow tract in 25 patients with tetralogy of Fallot and has proved satisfactory. While more difficult to handle than polyvinyl sponge, blood does not leak through the cloth itself in a heparinized patient. Experience with arterial replacement suggests that this material does not lead to aneurysm formation or perforation.

The temporary use of an encircling vest of compressed polyvinyl sponge to support the Teflon prosthesis until heparin was neutralized proved a most satisfactory expedient for the control of leaks between sutures. This was prepared and laid in place prior to the aortotomy. Following release of the aortic clamp the vest was closed by means of a few interrupted sutures of heavy silk that were later removed.

Left ventricular decompression by means of a catheter passed through the apex provided an immediately bloodless field while the aorta was open without

the use of a sucker tip passed through the aortotomy. In addition, this procedure prevents left ventricular distention following release of the aortic clamp and allows removal of air trapped in the left ventricle following the return of a satisfactory beat.[16]

SUMMARY

The clinical, hemodynamic, and operative findings in a patient undergoing correction of supravalvular aortic stenosis are described. The diagnosis was established preoperatively by left heart catheterization and selective angiocardiography. An oval-shaped patch of woven Teflon cloth (temporarily supported by a vest of polyvinyl sponge) used to widen the aortic lumen proved satisfactory.

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